Robert Ray
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July/59.

A

MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

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FIRST AMERICAN EDITION REVISED.

WITH

THREE HUNDRED AND NINETY-SEVEN ILLUSTRATIONS.

PHILADELPHIA:
BLANCHARD AND LEA.
1854.

PHILADELPHIA:

T. K. AND P. G. COLLINS, PRINTERS.

PUBLISHERS' ADVERTISEMENT.

In a work like the present, intended as a text-book for the student of pathology, accurate engravings of the various results of morbid action are of the greatest assistance. The publishers have, therefore, considered that the value of the work might be enhanced by increasing the number of illustrations, and, with this object, many wood-cuts, from the best authorities, have been introduced, increasing the number from 167, in the London edition, to 397 in this. In the list of illustrations, these additional cuts will be found distinguished by an asterisk (*) from those for which the authors are alone responsible. The selection of these wood-cuts has been made by a competent member of the profession, who has supervised the progress of the work through the press, with the view of securing an accurate reprint, and of correcting such errors as had escaped the attention of the authors. He has also added, at page 197, an account of the interesting microscopical observations of Dr. Donaldson, of Baltimore, on the characteristics of the true cancer-cell.

Philadelphia, November 1854.

PREFATORY NOTICE.

THE Authors of the present work have desired to lay before their professional brethren an outline of what is known in the domain of Pathological Anatomy. The absence of any original work in the English language, which embraces the whole subject, must be their apology for having made the attempt. They have sought to place before the reader a summary of ascertained facts, together with the opinions of the most eminent pathologists of this and other countries. They have regarded it as their duty to select, as far as possible, the best fruits from the harvest gathered by other laborers in this wide and interesting field. At the same time they have sought not to speak solely on the faith of others, even the highest authorities, but to investigate, as much as possible, for themselves, the correctness of the statements they adopted. They felt that, in some instances, better illustrations might have been obtained by borrowing from other works; but they were of opinion that the present manual would bear a stamp of greater truthfulness if the drawings were taken from objects seen and examined by themselves. They have therefore preferred (with few exceptions only) to use such illustrations as their own portfolios supplied. Although small drawings, in black and white, necessarily fail to give the important elements of size and color, almost essential to illustrations of Pathological Anatomy, the Authors hope that the masterly treatment of Mr. Bagg has achieved as much as could be done by wood engraving. They have divided the subject in the manner indicated in the Table of Contents, and are each individually responsible for the chapters which they have treated.

They conclude by expressing a hope that the vast extent of the subject, and of the material they had to deal with, will serve, in some measure, as an apology for the deficiencies which they are fully conscious of, and for which they ask the kind and lenient consideration of the Medical Profession.

C. HANDFIELD JONES,

EDWARD H. SIEVEKING.

LONDON, *August* 5, 1854.

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MANUAL

OF

PATHOLOGICAL ANATOMY.

CHAPTER I.

GENERAL OBSERVATIONS.

The object of General Pathology is to examine the various morbid processes which may occur in the human body, and to obtain, thereby, such an insight into their nature, that they may not be looked on as unknown entities, but that being comprehended as far as is possible themselves, the various effects they produce, the particular instances of their action, may be understood also.

The course we propose to follow is nearly that which Dr. Williams has adopted so successfully, in his work on *The Principles of Medicine.* We shall, therefore, describe, briefly: (I.) The morbid alterations of the several great functions. (II.) Those of the blood. (III.) Those of the various tissues, considered generally. (IV.) The superadded formations or growths, the so-called tumors. (V.) Parasitic beings, whether animal or vegetable.

Some general observations must, however, be premised. The term *Morbid Anatomy* hardly needs explanation; as ordinary anatomy implies the study of, and acquaintance with, the healthy structure, so morbid anatomy implies the same of diseased structure. The meaning of *Pathology* may be clearly conceived, by considering that of its twin sister, Physiology; as the latter imports the knowledge of the natural actions of healthy organs, so does the former that of the unnatural actions of diseased or disturbed organs. Physiology has her vital stimuli; Pathology her stimuli or excitants to unhealthy life. This expression brings us to notice a point which has been excellently illustrated by Prof. Simon. He remarks that many unnatural or diseased conditions are not really unnatural in themselves, but are the proper and necessary consequences of some cause or influence which has acted upon a healthy body. The state of skin which a severe burn produces is, certainly, very unnatural and diseased, but the inflammatory and exudative processes which have produced it are quite natural, under the

circumstances that have occurred; they are the proper reaction of a healthy organism to the unnatural stimulus of extreme heat, and are called forth in the same way as is the healthy flow of blood into a chilled part by the action of kindly warmth. So in a case of variola, the skin, covered all over with unsightly pustules, is in a very unnatural state; but it is not the eruption, nor the constitutional disturbance, neither, that is really unnatural, but the presence of a certain quantity of infectious matter in the blood, which, acting on a perfectly natural system, thus calls forth its expulsive efforts. If we slightly alter this perfectly natural state, as by premising vaccination, then the introduction of the variolous poison no longer produces the same morbid effects, and we say the system is protected. The fact is, the system is changed from its originally perfectly natural condition, and will no longer respond to the unnatural stimulus. We find, it may be, a portion of the brain so soft as to resemble cream, quite broken up and disorganized; but, we look further and find that the artery supplying it with blood has been plugged up or tied, and we then see that the *locus* of the disease was not really in the brain, but in the artery; it would have been abnormal had the brain, deprived of its supply of blood, retained its natural texture. Or, again, we see a person suffering from violent epileptic convulsions; but, he passes a large worm from the bowels, and the attacks cease: in the case of this individual, the convulsions were the natural expression of the unnatural irritation to which the brain was subjected. Many like instances might be mentioned, and they certainly show that disease is, very often, not to be regarded as a special entity of a peculiar, strange kind, but as the natural result of the endowments and qualities belonging to our bodily organs, when those organs are acted on by certain unnatural stimuli. Hence, we can better understand that many diseases have a regular and normal course, made up, so to speak, of a succession of necessary results, which, however, is liable to be disturbed by various extrinsic causes. For instance, a person has ague, the paroxysms occurring in regular succession; he takes quinine, and they diminish and disappear, the course of morbid action is interfered with and broken. Or, a child has hooping-cough, and the disease is proceeding in its usual course, but in consequence of exposure to cold, he is attacked with inflammation of the lungs, and the paroxysms characteristic of the disease are, to a great degree, interrupted; the hooping-cough is merged in the pneumonia. Or, again, a person has tubercular deposit in his lungs; the natural tendency of this is to soften, break down, and be expectorated, together with the involved tissue, while, as fresh deposits take place, more and more of the organ is destroyed; but, before this can happen, inflammation is set up to such an extent, in the surrounding tissue, that life is cut short, not by the effects of the tubercular destruction directly, but by the intercurrent inflammation.

The unnatural stimuli, provoking the succession of morbid actions, are often termed the *Exciting* causes of a disease; they may be adequate, when powerful of themselves, to produce their effect, or, may need the assistance of other causes, generally of a debilitating nature, which are called *Predisposing*.

But the question now occurs, whether all diseases are of the kind above mentioned, whether all can be regarded as the natural results of certain foreign injurious influences operating on the system. To this the answer, in the present state of pathology, must be, I think, certainly not. There are very many cases where we cannot point out any exciting cause of the existing malady, where it seems to have originated spontaneously, so far as we are able to judge. Of this kind are many instances of decay and degeneration of tissues, very many of malassimilation, or mal-secretion, hereditary diseases, and some congenital mal-formations. It may be that, as we advance in knowledge, we shall be able to include more and more of the latter class under the former; that as we obtain more acquaintance with the imponderable influences which are constantly in operation, we shall be able to refer to them as the causes of changes which now appear spontaneous, but from this we are far, at present, and must thoroughly recognize the two classes of disease which we have just described. These classes, however, are not (and natural groups never are) rigorously defined; there are numerous instances of an intermediate kind, such as those where a slight exciting cause calls into action an inherited predisposition. We must also notice another great division of diseases into two classes, viz: the Organic and Functional. Of the existence of the latter many of the best pathologists greatly doubt, that is to say, whether it be possible for the mechanism of an organ to be perfectly uninjured, at the time that its function is wrongly performed. Speaking in the strictest sense, and remembering the advances which have been made in detecting morbid alterations formerly unknown, as well as the amount of progress which we may yet look to make, it must certainly be allowed that it is quite possible that the division above mentioned is not founded in reality, and that all diseases are attended with organic change. But, when this is conceded, it remains still perfectly clear that there are not a few diseases, and some very severe, in which no organic alteration whatever can be detected; and it seems, further, a point of considerable practical importance that the student should be fully aware of this and alive to it. For the functional disease often nearly simulates the organic, or may succeed it, or exaggerate it, and the practitioner who is not carefully on the watch for the possible occurrence is very apt to be led astray. How often has dyspnœa, depending on disordered innervation, been treated as pneumonia, palpitation of similar origin considered as the result of serious cardiac disease, venesection been employed to relieve nervous headache, and so on! In this time of generally deficient power, and sensitive nervous systems, it behoves us especially to be on our guard against mistaking a functional for an organic disease. As in the preceding case, so in this, the two divisions which we recognize of diseases are not rigorously defined, they have each their marked exemplars, between which the intervening space is filled up with every possible grade. Functional disease may produce organic, either of the organ which itself affects, or of some other. Thus, epilepsy long continuing, produces morbid alteration of the cerebral structure; dyspepsia may be the cause of some unsightly cutaneous eruption. Organic diseases are much more often latent than functional. A man may have

serious heart disease, and think nothing about it, but he will be greatly alarmed if he suffers from an irritable palpitating organ. This fact, and the oft-repeated observation that post-mortem examination discovers no sufficient cause of death, testify, as well as the instances in which functional disease is itself fatal, to the reality and importance of functional disorder, either independent of, or out of all proportion to, organic alteration; and warn us that, necessary and philosophic as it is to investigate to the utmost the morbid changes of solids or fluids, and to endeavor to arrive by this way at a knowledge of the essential causes, and at sound indications of treatment, we must never overlook those grand plain intimations which nature gives of functional vigor or debility. Such considerations as these may be foreign to morbid anatomy; they are not inappropriate to pathology. What are called *idiosyncrasies* are unusual peculiarities of an individual system, in consequence of which it is affected in a different manner by some influences to that which is commonly experienced. Thus, some persons are attacked by asthma or bronchitis on inhaling the odor of hay; some are almost poisoned by taking the smallest dose of a mercurial. One at least has been mentioned (by Dr. Prout) who could not eat mutton in any form without being attacked by violent vomiting and diarrhœa. In such persons the qualities and endowments of one or more organs must be essentially different to those of the same parts in the vast majority of mankind. Yet there is not the least reason for supposing that, by any scrutiny, we could detect any structural difference, and they must, therefore, be deemed instances of aberring function.

One remarkable instance it seems worth while to adduce here, which affords an excellent illustration of the connection that may obtain between disordered function and alteration of structure. The Graafian vesicle in the ovary, instinct with a wonderful capacity of life, which only needs its appropriate stimulus to rouse it into that activity which issues in the production of another being, not unfrequently, as if affected by some strange and unnatural stimulus, proceeds to develop itself into a huge anomalous growth, utterly imperfect, and unlike what, under normal conditions, it should have produced, and yet exhibiting some traces such as are found in no other growth of its original destiny, by the formation of several of the natural tissues, skin, teeth, hair, nay, even brain, &c. Here, it seems impossible to recognize any other cause of the organic alteration beside the perversion or aberrance of a natural function or endowment. The term *Diathesis* is applied to a certain condition of the general system often inherited, which renders it especially liable to some particular form of disease; thus, we speak of the scrofulous or tubercular diathesis, of the gouty diathesis, and so on. If the diathesis or predisposition be strong, a slight exciting cause will be sufficient to induce the malady; if it be absent, no exciting cause may produce any effect. A diathesis may, therefore, be considered as a kind of special weakness. *Degeneration* of a part or tissue implies generally its slow and gradual conversion into some lower kind of structure less fitted for the purpose it has to fulfil, as when cartilage is converted into a kind of fibrous tissue; or it may imply the atrophy

and destruction of a part, as of the cortical structure of the kidney in Bright's disease. It is to be regarded in some measure as a local infirmity.—As in part a corollary and conclusion to the above remarks, I would subjoin the following great practical truth which daily experience presses on our attention, viz: that on the one hand there is a sound and healthy systemic life, of which all the organs are or ought to be possessed, and that, on the other hand, there often comes in its stead, either generally or locally, an unsound and unhealthy life, which leads to perverted morbid action, or to actual decay. The one is the "vis reparatrix," or "conservatrix Naturæ," withstanding and repelling morbid influences, sending a tide of life and vigor through the frame, striving to compensate for a casual loss or damage inflicted, and only sinking at last in the tranquil decline of a green old age. The other is that deathward taint, infecting and debilitating the even youthful system, opening wide the avenues to every casual morbid influence, increasing the power of every disease, and decreasing the capacity of resistance to it; and in fine, either embittering the years of a lingering existence, or cutting the span of life prematurely short. Well does the physician know and recognize in his patient the two states, different as the varieties of the latter may be. In the former, he confidently anticipates a successful result; in the latter, he prepares himself for a doubtful, difficult, perhaps only defensive warfare.

It was formerly a much debated question, whether diseases had their principal seat in the fluids or solids of the body; and each of the two opposed theories has at times been dominant. At the present day, we marvel how men could have adopted exclusively one view or the other, and refused to allow to each their share in the production of morbid phenomena. There can be no question, from known physical laws, that the blood must, in very many cases, be the first recipient of aeriform noxious matters, of all such miasmata as those of typhus, scarlatina, &c. The instant that these are drawn with the air into the lungs, they pass into the blood; for it is impossible that the gases contained in the blood shall not, according to the law of heterogeneous attraction, be exchanged in part for those which are diffused in the air-cells and cavities of the lungs. As little doubt can there be that the blood, as it is the first to receive, so it is also the first to be modified and altered from its healthy composition by the inhaled miasm. In the great class of inflammations, the affection of the tissue and of the blood must proceed *pari passu:* so intimately is the blood concerned in every stage of the process, that it may almost be said both blood and tissue are alike the seat of the disease; but the latter manifestly has the initiative. Rheumatic and gouty inflammations must, however, be excepted, in which the blood is certainly the primary seat of morbid alteration. Scrofulous disease, in all probability, commences in the blood, and produces in it a change, of the nature of which we are ignorant, but which issues in the deposit of a peculiar matter in various localities. In diseases arising from excess in eating and drinking, or from unwholesome aliment, the blood in some cases, and the alimentary canal in others, may be primarily affected. When we consider what the processes of nutrition and secretion imply, how the blood is a vast

laboratory, in which some secretions are actually prepared, and the materials of others; how it conveys to each part the nutriment that is appropriate to it, and receives back in return principles more or less effete; how continually it is receiving supplies of new matter from without, and undergoing depuration by various appointed emunctories; in short, if we consider how thoroughly the different solid and fluid parts of the frame are correlated, and mutually dependent, we shall perceive most clearly that it is far more important to be fully aware of the extreme liability, nay, necessity, of the solids to be affected by the fluids, and the fluids by the solids, and that thus the disorder of one part may be the exponent of the error of another, than to attempt an almost impossible definition of the exact origin and site of a disease.

FUNCTIONAL DERANGEMENT.

We now proceed to consider the morbid alterations which are observed in some of the great functions or endowments belonging to some of the most important tissues of the animal frame. These have been termed by Dr. Williams Primary Elements of Disease; and it seems especially desirable to obtain as exact an acquaintance with them as is possible, because in these instances morbid action presents itself in its simplest and least complicated form. In almost all diseases two or more of these are variously combined, and success in treatment depends very much on the due appreciation of the several existing elements in each case, and of the degree in which one or other predominates. In a common case of irritative dyspepsia, for instance, the sensibility of the nerves, the tonicity of the vessels, and the secreting action of the follicles, are all variously affected, and all react upon and aggravate each other. The judicious practitioner will bear in mind the existence of all, and will endeavor to apportion his remedies according to the predominance of one or other.

We shall mention first disordered states of *Contractility*, including under this head the modification of it termed *Tonicity*. This is the property of muscular fibre, both of the striped and unstriped varieties, though it is manifested differently in them. The contraction of the former immediately takes place on the application of a stimulus, and soon subsides; that of the latter comes on more gradually, and is of longer endurance. It has been supposed by some that the power or quality was not resident in the contractile tissue, but in the spinal cord, from which it was conveyed by the nerves to the muscles; but this view seems quite contradicted by observation and by analogy. It is, however, perfectly true that contractility is almost always called into play by the instrumentality of the nervous system, and that most of its apparent disorders have really their seat in the nervous system. Still, we believe that there are disordered conditions of contractility itself, though these may sometimes have been originally produced by disordered innervation. The principal one of these is a condition which may be termed unnatural mobility. The tissue is so irritable that it is thrown into action on the least stimulus, or even seems almost to contract without a stimulus.

At the same time the contraction is often feeble and imperfect, hurried and irregular. It reminds one forcibly of the mental condition of an anxious, restless, incapable person. This unnatural mobility is often observed in the hearts of anemic patients, sometimes in those of persons who have undergone severe exertion without proper previous training. It is also exemplified in the irritable bladder, which will not contain the least quantity of urine, in that state of the intestinal canal which gives rise to what is called lientery, and in some conditions of the stomach, where every particle of food or liquid that is taken is immediately rejected. It seems doubtful whether contractility is ever so exaggerated as to amount to a morbid state, that is to say, contractility regarded in its power of action, not merely in its readiness of being excited. We might have feared that when muscles became enlarged, and their vigor increased by frequent exercise, the osseous levers which they move, and which give them attachment, might have proved inadequate to resist the increased strain; but experience shows us the beautiful adaptation which provides that, as the muscle hypertrophies and its force increases, the bone also enlarges and strengthens in an equal ratio. It may be that in some cases of spasm or cramp the contractility itself of the part is excessive, and that it is not only the nervous system that is at fault.

Instances on the other hand are numerous, in which contractility is defective. The weakness which we feel on first leaving a sick bed, when we can scarcely raise our limbs, depends on defective contractility of the muscles, occasioned by long disuse. The same may be the result of paralysis, of rheumatic inflammation, of the poisonous action of lead, of tobacco, sulphuretted hydrogen, &c. Impaired contractility is a very frequent cause of habitual constipation, which may then be best treated, as in a case recently under our care, by tonics. The failure of repeated purgatives in this instance also exemplifies the truth, that, after violent excitement, contractility loses still more its energy, and is still more difficult to be called forth. The failure of contractile power in many cases, but certainly not in all, is connected with an actual change in the structure of the fibre. Of course, if this has degenerated, it cannot discharge its function properly, but in other cases the failure of the function probably precedes the degeneration.

Tonicity is observed in the striped muscular fibre, as well as in all the varieties of the organic; it is what Mr. Bowman has denominated Passive, in opposition to Active contractility; the latter being the response of a fibre to a stimulus, the former a constant state of tension, or approximation of all the points of the fibre throughout its length. There is no real difference between tonicity and passive contractility, yet it may be stated that the former designation is most applicable to the contractility which is manifested by the coats of the bloodvessels, and the skin. Heat and cold are the influences which have most effect upon tonicity, the former producing marked relaxation, the latter contraction of the tissues which possess it. The possession of this property, by the bloodvessels, is of the very greatest importance; it is intimately concerned in many morbid phenomena, and notably in those of inflammation. When we place our finger on a small, hard, wiry-feeling pulse,

such as exists in the outset of peritonitis, and other membranous inflammations, we recognize a condition of the artery in which its coats are tensely contracted on the stream of blood passing through it, and form such a firm cylinder as not to yield to the pressure of the finger. Here tonicity is in excess. The rigid state of the walls of the arteries prevents their yielding to the distensive force of the wave of blood thrown in by each contraction of the heart, and hence there is not the usual interval between the impulse at the chest and the pulse in the limbs. In persons of a sanguine temperament, tonicity is probably in every part somewhat excessive, the muscles are more rigid, and the pulse more firm, than is consistent with the most perfect health; while in persons of a lymphatic temperament the same quality is deficient in a corresponding degree. When tonicity is naturally somewhat excessive, a cold, dry atmosphere, or an easterly wind, may cause considerable discomfort and disturbance of the system. The superficial vessels and the integuments are so constringed, that the blood is repelled inwards in undue quantity, and the exhaling function of the skin materially interfered with. On the contrary, where tonicity is naturally defective, the very same influences may be of decided benefit. A case has come within the knowledge of the writer, in which the tonicity of the cerebral vessels became greatly impaired in consequence of enfeebled health, and much mental strain. To such an extent had this proceeded, that sleep was very much disturbed, and rest prevented; as immediately on lying down blood was transmitted in undue quantity to the brain, unrestrained by the toneless vessels, which were felt pulsating violently. The relief experienced in this case on the setting in of cold weather was most marked, and was clearly produced by the diminished temperature having aroused the tonicity of the weakened vessels. That distressing affection which not remotely simulates aneurism, an atonic condition of the abdominal aorta, is another instance of the same kind. It is clear that an atonic state of the vessels must predispose extremely to local congestions, which may often advance to asthenic inflammations; it must also be a frequent cause of a varicose state of the veins. In those alarming, but happily rare cases, where the slightest wound occasions considerable hemorrhage, which can scarcely be restrained, it seems most probable that the tonicity of the vessels is chiefly in fault, that they do not contract as they should when cut across, and thus close the bleeding orifices. The effect of cold in restraining hemorrhage, and of heat in promoting it, is matter of common observation, and is of course produced by their action on the coats of the vessels. The state of the pulse is an excellent indication of defective, as it is of increased tonicity; the soft, yielding, large vessel has evidently its coats in a state of relaxation, and the wave of blood in it is not felt till some space after the impulse at the chest. Nervous influence is evidently capable of arousing or depressing tonicity; but it is equally clear that the quality itself exists in very various degrees. The loss of tonicity in the vessels leading to an inflamed part, we shall hereafter see to be an important circumstance in the process of inflammation.

From the examination of that great system which is the seat of contractile power, we proceed to that which is the seat of nervous power;

and we shall first consider its disorders with regard to its faculty of receiving impressions and converting them into sensations. This power is termed Sensibility; it may be morbidly increased, or diminished, or perverted; and these alterations may be either general or local. When sensibility is generally excessive, the indications of it are so plain, that it is almost needless to enumerate them. The sufferer may be compared to a sensitive plant—shrinking from every touch. Every pain or ache seems prodigiously magnified in intensity, and is described in hyperbolic language. The important points to bear in mind in dealing with such cases are: (1), that real disease may coexist with this hypersensitiveness, and that we must not, therefore, too hastily ascribe all "to the nerves;" and (2), that if such real disease do exist, some of its symptoms will probably be greatly intensified to the patient's sensations, so that we might easily be led to think the disease more serious than it really is. The condition now mentioned is essentially chronic; and is to be distinguished from that state which exists in cases of inflammation of the brain, or determination of blood thither, or an irritation of the organ from any cause. In these there is also intolerance of light and sound, and any jar or movement is painful. This condition, however, is never of long continuance, and is often succeeded by an opposite state, and is evidently dependent upon inflammatory excitement. Delirium tremens is not properly an instance of increased general sensibility, as it does not appear that the nerves are concerned in exciting the disorder of the sensorium. It seems, however, so far to belong to the morbid state we are now considering, in that it consists, certainly to a great degree, of an unduly excited state of the sensorial centre, the excitement, however, being dependent rather on immaterial than on physical impressions. The known exciting causes of delirium tremens are also just those which are likely to produce excessive sensibility; they are such as irritate and excite the nervous system for a length of time, succeeded by such as occasion general debility. This affection, therefore, with its occasional tendency towards inflammatory excitement which it sometimes presents, seems to hold an intermediate place between the common chronic habitual hyperæsthesia, and that excessive intensity of the function which is seen in phrenitis, &c. Sensibility may be increased locally—a part may be so tender as to be almost unable to bear the lightest contact. This depends sometimes on an inflammatory condition of the part, sometimes on a simply altered state of its innervation. The illustrations of the first are of constant occurrence; those of the second are furnished by the instances of what is called irritable breast, testicle, or uterus. The intolerance of light, which is so marked a feature of scrofulous ophthalmia, shows a condition of the retina which must be regarded as one of hyperæsthesia, not dependent on inflammatory action. It is very remarkable that the internal organs, which, in their healthy life, are devoid of common sensibility, should, when affected by disease, become so acutely sensitive. Who knows aught of what is going on in his stomach, while it is digesting healthfully? or how his gall-bladder or intestines or ureters are acting on, and transmitting their contents? But let disease arise in these parts, and then their actions become attended with pain, some-

times so severe that the like is seldom experienced. We are able, in some measure, to account for the fact, by remembering that the sympathetic nerves, which supply the intestines, &c., contain numerous cerebro-spinal fibres, and also that the nerves, in their course, pass through numerous ganglia, which probably serve, under ordinary circumstances, as centres of nervous influence, beyond which the impressions are not conveyed. Diminished sensibility is not unfrequently observed as the result of stupor, or coma, in whatever way induced. It is necessarily consequent on division of the spinal cord, in all parts supplied with nerves below the seat of the lesion; and it often occurs partially in apoplectic or paralytic attacks. In these, however, it is rarely so complete or so persistent as the loss of motion. The retina not unfrequently loses its sensibility, either for a time or permanently, without exhibiting any trace of disorganization; and there are analogous instances in which deafness, more or less complete, results from paralysis of the auditory nerve. The application of cold is a powerful means of diminishing sensibility. Of this, we have a familiar instance in the numbed condition of the fingers which is so frequent in cold weather. In this instance, the nerves lose their sensibility, in consequence of their being deprived of their usual supply of blood; the part is anemic, as well as numb; and this arrest of the circulation again depends on the action of cold upon the tonicity of the arteries, which become so contracted that they no longer transmit a free current of blood. Here we have a good illustration of the dependence of the several parts and functions one upon another. It is, however, most probable, or almost certain, that cold directly tends to diminish sensibility, by its action upon the nerves themselves. We have constant opportunities of observing how confinement to warm rooms tends to induce a state of generally increased sensibility; and how this condition is corrected and removed by a free exposure to the inclemencies of the weather. Sensibility may be perverted in many various ways. Persons suffering under cerebral affections, either functional or organic, experience sometimes peculiar sensations in different parts of their bodies. These may resemble the crawling of insects over the surface (formication), pricking with pins and needles, tingling, &c. Pruritus, and all the varieties of itching, must generally be referred to a peculiar alteration of sensibility in the nerves, distinct from that which is produced in them by common inflammation. In the severest forms of this affection, no inflammation at all is present. It is interesting to observe that itching may be dependent simply on a disordered crasis of the blood, or the presence of some unnatural element in this fluid. Thus in jaundice this symptom is frequently observed, and is doubtless occasioned by some biliary constituent being absorbed from the liver, and carried along with the blood. Pain seems to be properly included also under the head of perverted sensibility, as it is certainly something more than simply exaltation of this endowment. On this ground we may refer to neuralgia, and especially to the affection called tic douloureux, as affording the extremest instance of perverted and also exalted sensibility. The affected part may be sometimes the peripheral; more often, probably, the central. However, in neither case does there appear to be any alteration of structure that

could account for the disease.[1] This instance, and that of pruritus, are especially worthy of notice. They seem to afford us strongly-marked examples of extreme disturbance of a single function, without any complications, and unattended by any discoverable lesion of structure. Another circumstance worthy of remark, with respect to altered sensibility, is that the symptom does not always manifest itself in the part which is the seat of irritation, but in some other, at a distance. Thus, severe headache is occasioned in some persons by taking some article of food which offends the stomach. Pain in the knee is a well-known symptom of disease of the hip joint; and pain at the extremity of the penis, of a stone in the bladder. No satisfactory explanation of this fact has yet been given; but it seems most probable that the impression transmitted to the centre, there affects, in some unknown way, the contiguous extremity of the nerve of the part to which the sensation is referred.

Another function of the nervous system, of its central organ, is that of Voluntary motion, and this also is subject to morbid alteration. In mania and delirium, the power is sometimes extraordinarily intensified, so that a naturally weak person may resist the force of several strong men. Great mental energy and powerful emotions also increase the power to a great degree. The brain in such conditions may be compared to a highly charged and constantly acting electric battery. On the other hand, debilitating and depressing causes, congestion, narcotism, or stupor, impair the voluntary power, as also sudden alarm, or great anxiety. To speak of the paralyzing effect of terror is scarcely a metaphor. Instances of perversion of this faculty are found in various strange affections, more or less allied to hysteria, such as the tarantula dance, certain religious ecstasies, and the cataleptic state. Partial defect of voluntary power is very commonly observed as the result of interruption of the transmission of nervous influence to the part. Thus, if the dynamic mechanism of the centre be damaged by apoplexy, or the texture of the cord be destroyed, or a nerve subjected to atrophic pressure, paralysis more or less complete of the part must take place. But there are certain cases in which there is no reason to imagine that any disease of the nervous system exists, yet affected with more or less complete paralysis of some part. To such the term hysterial paralysis is applied; they are rather diseases of the mind or emotions than of any part of the bodily frame. It is very important to be aware of the existence of such diseased states, both for the sake of avoiding errors in treatment, and also that we may estimate aright the wonderful influence which our immaterial is capable of exerting upon our material organism.

That property of the spinal cord and its prolongation upwards within the cranium, by virtue of which an impression communicated from an internal or external surface occasions a motor impulse to be conveyed outwards along corresponding nerves which are distributed to certain muscles, is liable to be morbidly affected, and thus to become the cause

[1] Dr. Romberg, however, has given the details of a case in which the most severe facial neuralgia was caused by disease of the origin of the fifth pair.

of some of our gravest and most fearful maladies. The conception we have to form of this power is the following: The gray matter contained in the spinal cord, medulla oblongata, and associated cranial gangliform masses, constitutes a great system, which Dr. Marshall Hall terms the true spinal, as distinguished from the cerebral, the latter consisting especially of the hemispherical ganglia. Now, whereas voluntary motion is produced by the will operating a certain change in the gray matter of the hemispheres, which change communicates an impulse to the interwoven nervous filaments, to be conveyed by them to the point of origin of the nerve to be affected, and so along this to the muscles, the mode in which reflex action takes place is different. A change, generating an impulse, is also produced in the gray matter; but it is not in the gray matter of the hemispheres, but in that of some part of the spinal centre; and the excitor of the change is not the immaterial mind, but a physical change in some centripetal nerve, induced by some stimulus applied to its extremity, and propagated along it to the point where it is implanted in the dynamic centre. The motor impulse thus excited may affect some adjacent muscular nerve, or be communicated to others implanted in some other part of the spinal centre. The use of this power is clearly for the constant maintenance of certain muscular actions, necessary to life, and the protection of important parts, which could not have been left to the uncertain agency of volition. In a certain part of this centre, that termed the mesocephale, emotional impulses appear to be particularly resident, and to be called forth by the nerves herein implanted. In the natural condition, scarce a single reflex act takes place that is not attended with sensation. The same impression that determines the movement, takes effect also as a sensation; but if the communication with the hemispheres be cut off, or consciousness be suspended, the reflex actions take place quite independent of sensation. So also the will has power over many of the muscles, which are under the sway of reflex action; but if volition be cut off, the actions are still carried on, nay, they cannot be prevented, by any exercise of the will. Reflex action may thus be regarded as an "imperium in imperio," and as absolutely dominant within its own territory. To the power of reacting upon motor nerves—*i. e.* of generating an impulse within itself, on the reception of a centrifugal impression, which resides in the spinal dynamic matter—Dr. Todd has given the name of polarity. The term is an expressive and convenient one, and we shall adopt it. Now this power is capable of being enormously increased, both as regards its susceptibility of being aroused by stimuli, and also as to the extent in which it is exerted, and the potency of its action. Under ordinary circumstances, the voluntary muscles are, speaking generally, exempt from its influence; but in some diseases they are so overmastered and controlled by it, that the will is almost powerless over them. At the same time the polar susceptibility is so greatly increased, that everything, even a breath of air, operates as a stimulus, and the dynamic matter, like a powerful battery, discharges its impulses with terrible force through the motor nerves upon the muscles. This condition of the spinal centre can be produced artificially by the administration of certain poisons, especially strychnine, which seems, indeed, when

given in a sufficiently large dose, to exalt the polarity immediately on its reaching the cord. In Mr. Blake's experiments, twelve or sixteen seconds only elapsed from the time when the poison was injected into a vein, until convulsions commenced, the proof of the cord having been already affected. This fact, as well as results of Dr. Todd's examination of the spinal cords of animals who died from the effects of strychnine, show conclusively that inflammation has no share in the production of the phenomena, and that the essence of the malady is purely an unnatural exaltation of the normal polarity. Tetanus, whether idiopathic or traumatic, is precisely identical with the condition induced by strychnine, and, like it, proves fatal, either by utter general exhaustion, or by unrelaxing contraction of the respiratory muscles, and consequent suffocation. Chorea is an analogous state, in which volition and the spinal polarity seem to maintain a doubtful contest for dominion over the muscles, which are usually subject to the will. Hysterical convulsions are often dependent on some cause of irritation in the intestines. The impressions conveyed from hence excite the unduly susceptible emotional centre; and while they produce the peculiar psychical phenomena, are also reflected upon the muscles in impulses to convulsive action. In eccentric epilepsy, the mode of causation of the attacks is essentially similar; but it is probable that a different part of the spinal centre is affected. It is possible that, in centric epilepsy, the attacks depend on a periodical exaltation of the polarity of the centre, in consequence of which, as in tetanus, the slightest impressions excite the most powerful motor impulses. In early infancy, the cerebral system has not acquired that predominance over the spinal which it is subsequently to attain; and, moreover, the process of dentition, as well as the delicacy and susceptibility to disorder of the intestinal mucous membrane, seem both to excite the polarity of the cord, and to furnish causes of irritation, which, acting on the excited centre, occasion the convulsions which are so common a manifestation of nervous disorder in children. One very frequent and important instance of this kind of disorder deserves particular notice. Delicate and weakly children not uncommonly are affected with spasmodic contraction of the glottis, more or less complete. In its slighter degrees, this produces a kind of crowing sound, from the rush of air passing through a narrowed orifice. In its extreme degree, the glottis is completely closed; no air can be drawn into the chest, and the countenance turns livid. In this state death may occur, or the spasm may suddenly relax, inspiration be effected, and the imminent peril escaped. The system, however, remains in the same state of polar tension, and life is in serious danger from the frequent recurrence of such attacks, any one of which may prove fatal. How obscure would be the pathology of this disease, without the clear light which Dr. Marshall Hall's discovery has shed upon the subject! He points out how it originates in the trifacial nerve in teething, in the pneumogastric in over or improperly fed infants, in the spinal nerves, in cases of constipation, intestinal disorder, or catharsis. These act through the medium of the spinal centre, from which motor impulses are reflected upon the inferior laryngeal, and, in some cases, perhaps, on the intercostals and phrenic nerves also. It is worth

remarking, that general debility, which increases sensibility, seems also to favor the increase of polarity. The condition of grave inflammation is not that which seems most to promote the occurrence of reflex phenomena. The cough, in a case of pneumonia, or pleurisy, is often not nearly so distressing as that which follows upon some trifling catarrh, when some flake of acrid, irritating mucus, is lodged in the follicles of the upper part of the trachea, or larynx, and not being easily detached, keeps up a perpetual excitement of the incident nerves, which is reflected upon the motor to the expiratory muscles in ceaseless impulses to cough. Many other instances might be referred to, but enough has been said to show that the idea of polarity and its disorders should never be absent from the mind of the rational physician. Instances of defective polarity are not unfrequent, though less striking than those of increased. Such a state is observed in some cases of fever, where the blood seems incapable of maintaining the natural power of the medulla, in consequence of which the impressions conveyed by the pneumogastric are feebly responded to, and require the occasional assistance of voluntary inspiratory efforts. The same is observed in some diseases affecting the head, and hence this peculiar kind of respiration has been called cerebral. The following quotation, from Dr. Williams's work, contains all that can be said upon the subject: "A failure of this function, similar in kind, but less in degree, is exhibited in all states of extreme debility, whether from excessive fatigue or excitement, or from directly depressing and sedative influences, as in adynamic fevers. A person in this state is *too weak to sleep*, for the medulla, partaking of the general exhaustion, cannot maintain the respiration without assistance from voluntary efforts. Hence the feeling of oppression and the frequent sighing, which banish all repose; or if sleep do occur, it is disturbed by startings and fearful dreams, occasioned by the painful sensations of imperfect breathing.

Derangements of Nutrition and Secretion certainly constitute primary elements of disease. Of the former, we shall speak particularly when we describe the various degenerations that affect the different organs. The latter will be considered in detail under the head of the several secreting organs and their respective products. We shall now only offer a few general remarks on these derangements and their effects. Nutrition and secretion are evidently in great measure processes of identical nature. The chief difference is, that in the latter a considerable part of the nutrient supply is conveyed out of the organ by tubular channels, more or less altered from the form in which it exuded from the bloodvessels. The processes of secretion in the different instances presented by the system are not all exactly alike, the pulmonary secretion, and the urinary in part, seem to pre-exist ready formed in the blood; the biliary and the gastric, as well as the seminal, must be formed in their respective glands. It seems probable, however, that in all cases an appropriate blastema is requisite for the due performance of the function of the organ, and that this contains either the secretion ready formed, or principles which are in course of change, or ready to change into it. Part of the secreting process is, therefore, accomplished in the blood, part in the several glandular

organs; and the proportion which these bear to each other varies in different instances, and perhaps to some extent in the same. In nutrition, there seems scarce any reason to believe that the tissues produce any very considerable change on the blastema supplied to them, the sarcous elements of muscle are but slight modifications of albumen, the bones receive phosphate of lime ready formed in the blood, the nervous matter is chiefly a compound of oil and albumen, the various pigments are probably modifications of that of the blood-globules; and even those which depart most widely from the composition of the blood, the various gelatin-yielding tissues, may fairly be regarded as having no very distant connection with the Protein compounds. Now it is a point of prime importance to remark, that, in both the nutritive and secretive processes, the failure or imperfect performance of the function in any one instance produces an injurious effect on the circulating blood. For the nutrition of a part does not imply merely the withdrawing of a certain amount of Liq. Sanguinis, and its appropriation to that part, but the separation of a fluid differing *qualitatively* more or less from the general current, in consequence of which certain elements are retained in, and become, therefore, more abundant in the blood. Now if the proper selection of material does not take place in the maintenance and repair of different tissues, it is manifest that the composition of the blood must be altered. Again, nutrition involves the decay of tissues, and the reabsorption into the blood of their effete parts; from which it is clear that, on the due performance of what Dr. Prout calls secondary destructive assimilation, the healthy condition of the blood is in part dependent. No doubt there are physiological limits, within which the nutrition of different parts may vary; but if these are exceeded, disorder, first of the blood, and subsequently of other parts, must ensue. It is difficult to point out positive examples of disease arising from such causes, but it seems right to refer to them, as they may probably lie at the bottom of many obscure and ill-defined morbid states. With respect to the secretive processes, we have familiar instances of their disorder producing injurious effects on the blood, and through it, upon other parts. If the liver become sluggish in its action, and bile is not properly excreted, the countenance betrays by its tinge the unnatural state of the blood, and the loss of appetite and headache testify that the stomach and the brain are secondarily affected. If the texture of the kidney be spoiled, and the secretion of urine in consequence be seriously interfered with, the urea is retained in the blood, and this fluid becomes thus so altered in its composition, that the red globules are no longer properly developed, and the patient presents a sallow, anemic aspect, while at the same time inflammations are exceedingly apt to arise in various parts, owing to the disordered nutrition induced by the unhealthy blood. If the secretion of a gland be greatly increased, though of perfectly natural composition in itself, this increased outflow becomes a drain upon the system, and thus a cause of debility. Diabetes may be referred to in illustration of this, as, although an unnatural substance, sugar is added to the urine, yet its own composition is not materially altered. Another very striking instance is afforded by cases of asthenia lactantium, the continued drain

from the mammary glands exhausting the frame and all the vital energies in a fearful manner. Secretions excessive in quantity, and more or less unnatural, also produce great debility; of this, we have frequent examples in profuse diarrhœa or leucorrhœa. The material of these fluids is of course so much withdrawn from the circulation. Unnatural secretions often produce irritation and disturbance of parts with which they come into contact. Thus acrid bile produces severe diarrhœa; diarrhœal and leucorrheal discharges often excoriate the integument around their respective outlets, highly acid urine causes a sensation of scalding in the urethra, or even may give rise to attacks resembling nephritic colic. Deficient quantity and disordered quality of a secretion, often go together; thus scanty urine is generally morbid in some other respect; the opposite condition, however, is quite as frequent, and the secretion, though plentiful, is very unnatural. Of the latter condition we have examples in debilitated persons who pass large quantities of pale, alkaline urine, containing triple phosphate. The former state is constantly observed in the commencement of various febrile affections. The nervous system has a considerable influence over the various secretions. Great agitation has been known to cause a mother's milk to assume a poisonous quality, or such, at least, as to occasion in a few minutes the death of the infant. A similar cause has produced jaundice rapidly in some persons, and in others a bilious diarrhœa. After an hysterical fit, a large flow of pale, almost aqueous, urine is passed. A flow of tears is the natural effect of the passion of grief, and a flow of saliva of the expectation of a meal. Appetite is immediately destroyed, *i. e.* the secretion of gastric juice arrested, by sudden distressing intelligence. The lessons which these facts convey can scarcely be too much appreciated. They show us that we must never forget the wonderful but intimate connection that exists between our material and immaterial part, and that it is fruitless to strive against the incessant influence of a down-weighed or wounded spirit by doses of drugs. These cannot "cleanse the stuffed bosom of that perilous stuff which weighs upon the heart." Instances are occasionally met with, in which some secretion is manifestly unnatural, and yet there is no constitutional disturbance. It appears as if some morbid matter were carried off by this channel, the removal of which left the system in health. Of this kind are cases of fetid secretion from the feet, some of oxalate of lime in the urine, and perhaps the naturally foul breath which is habitual to some persons. It is very probable that several disorders, among which may be particularly mentioned gout and rheumatism, essentially depend, partly on a mal-performance of that part of the function of secretion which takes place in the blood, and partly upon defective elimination; so that various effete matters, not undergoing those oxidating changes which they normally should, and being, instead, partly converted into other more noxious and unnatural principles, circulate in the blood for some time, producing general uneasiness and malaise, and, sooner or later, break out in an eruption of morbid matter, by the skin, or some other emunctory. The gouty paroxysm, with its foregoing ill-health, is the παραδειγμα of this condition. It is also illustrated in rheumatism, and more or less in

other morbid states of the system, to which the appropriate name of Excrementitious Plethora has been applied. Dr. Williams observes, that he has often found purpura connected with hepatic congestion and imperfect excretion of bile, and most effectually removed by remedies which promote the restoration of the proper secretion. It is not unfrequently seen that the sudden arrest of a secretion, though it be a morbid one, which has continued long, and produced a considerable drain on the system, is attended with serious, nay, even fatal effects. These probably depend on the establishment of a condition of plethora, not, indeed, such, as under ordinary circumstances would deserve the name, but which is felt as such by the debilitated, and perhaps sensitive, system. When this state exists, local congestions are very apt to occur, and may end in fatal extravasation of blood in the brain, if that be the part affected. If the natural secretion of a gland be in any way greatly diminished, a state of congestion of the organ is very apt to follow; the converse occurrence also is often observed, and it is not by any means always to be discerned clearly which of the two is to be regarded as the cause, and which as the effect. The temperature of a part whose natural secretion is arrested, is almost always higher than natural. No more marked instance can be mentioned of this than the skin in many fevers. Remedial means, which diminish the quantity of blood in a congested part, often restore or increase the secretion which had been interrupted; and conformably to this, we often observe in cases of profuse abnormal secretion, that the surface from which the flux takes place, instead of being red with blood, is unnaturally pale; the contents of the vessels seem to be drained away as fast as they arrive; so that one is almost reminded of the old theory of exhalant arteries with open mouths.

CHAPTER II.

MORBID STATES OF THE BLOOD.

THE saying attributed to Cuvier, "Le sang est chair coulant," expresses very fairly the relations that subsist between the nutrient circulating fluid and the solid tissues. As we have already remarked, a change in the former involves almost necessarily a change in the latter, and each of the vital actions of the latter exerts some influence on the former. It is manifest, therefore, that an acquaintance with the healthy properties and morbid alterations of the blood is absolutely essential to a correct study of the phenomena wrought by disease in the solid parts. But the blood itself is a very compound thing, not only as regards the number of different matters it contains, organic and inorganic, not only as regards the manifold additions which it receives from without in the way of nutriment, and those which are poured back into it as the effete residues of tissues, but also as regards its own morphological condition being made up of solid and of fluid parts, of organized particles floating in an organic liquid. In diseased states of the blood these several parts, or some of their constituents, may be separately affected, and it seems, therefore, desirable on this account, as well as for the sake of greater precision, to examine first the individual component elements of this fluid with reference to their pathological changes, and afterwards to consider the diseases of the blood taken as a whole. Regarding the blood *per se*, this inquiry would rather belong to special than to general Pathology, but we include it under the latter head on account of the manifest general relations of the blood.

RED AND WHITE CORPUSCLES.

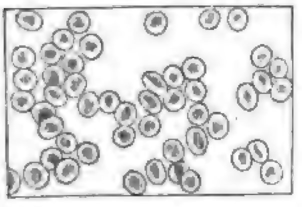
The circulating fluid consists, as we know, of two kinds of organized particles, floating in a transparent slightly yellow fluid. The organized particles are the red globules, preponderating immensely in number, and giving to the fluid its characteristic color, and the white or colorless, which in the healthy state occur somewhat "few and far between," and impart to the general mass no distinguishable quality.

Becquerel and Rodier.

	In Males.	In Females.
Corpuscles, red and white	141.1	127.2
Water	779.0	791.1
Fibrin	2.2	2.2
Albumen	69.4	70.5
Fat, extractive, salts	8.4	9.0
	1000.	1000.

The red globules are vesicles having a distinct homogeneous envelop, which incloses a central mass of whitish albuminous matter, and a surrounding film of red fluid. Their form is circular and biconcave, they present therefore cupped surfaces and an 8-shaped edge. They readily distend themselves by endosmosis with thinner fluid and assume a spherical shape. On sudden pressure being applied, and sometimes

Fig. 1.



Blood-corpuscles, magnified 400 diameters.

Fig. 2.

White Corpuscles of the Blood, magnified 400 diameters.

from other causes, they assume a kind of crenate form, conveying the idea that the central mass, the so-called globulin, is broken up into a number of granules, pushing irregularly outwards the homogeneous envelop. The white corpuscles are much larger, nearly double the size of the colored ones; they are spherical, and may often be seen when a drop of blood is examined under the microscope, remaining motionless and fixed in the field, while the red globules are hurried along on all sides round them by the currents which are created in the fluid by capillary attraction. No true nucleus can be seen in the white corpuscles;[1] they seem to consist of a semi-solid mass of albuminous matter, inclosed within a delicate envelop. Their function is not certainly known; by some physiologists they are regarded as constituting, together with the similar corpuscles in the lymph and chyle, transitional forms in the development of the red particles. By others, they are supposed to be the agent in the formation of the fibrin, which they elaborate out of the albumen, imparting to it its peculiar coagulating and so-called plastic properties. The use of the red globules is in all probability connected with the function of respiration; this seems well-nigh demonstrated by the facts of comparative anatomy, and by the marked changes which they undergo in the course of the circulation. Liebig has expressed the opinion that they are the carriers of oxygen to the various tissues, or, more exactly, the iron which they contain; this element in its protoxide form receiving in the lungs another atom of oxygen, and thereby passing into the state of peroxide, while in the course of the circulation it again parts with the atom of oxygen in exchange for one of carbonic acid, and thus passes into the state of protocarbonate. This view may be in part true, but is doubtless too exclusive; there is as much reason to suppose the Liquor Sanguinis to be the vehicle of the oxygen and carbonic acid, as the red corpuscles.

[1] Acetic acid brings into view, or produces one, two, or three granules similar to those contained in the pus cell.

Mr. Wharton Jones believes that the red globules elaborate the fibrin, or rather are transformed into it, bursting and dissolving like secreting cells. Various plausible arguments have been advanced in support of this view, which was first propounded by Dr. Simon, of Berlin, and is adopted also by Wagner and Henle; but it does not appear to the writer that it can be considered at all established. Neither on the other hand can Dr. Carpenter's be considered proved, though the preponderance of testimony seems on the whole to be in its favor; nor is there, in short, any positive cause to forbid us to believe that the truth may lie intermediate, and that the production of fibrin may be one result of the general cell growth taking place in the blood. With respect to the red globules, however, this is certainly proved, that their amount is, *ceteris paribus*, proportionate to the vigor, health, and strength of the individual. In the examinations made by MM. Andral, Gavarret, and Delafond, of the blood of various animals, it was constantly observed that those which possessed most strength and vigor, and were generally the finest specimens of the race, gave the highest figures in a series showing the relative amounts of globules; while those that were debilitated and poor showed a corresponding deficiency in this particular. Also, when the breed of a species was improved by crossing it with another, there was a corresponding increase in the quantity of red particles. In the human subject, the comparison of the general vigor and activity of the sanguine temperament, both as regards body and mind, with the sluggishness and dulness of the lymphatic temperament, or with the languor and debility of the anemic patient, show that the same rule holds good. In Prevost's and Dumas's experiments, animals near death from loss of blood, were recovered by an injection into their veins of a mixture of red particles and serum. The serum alone had not this effect. The proportion of red globules *dried* to 1000 parts of blood, is in healthy males estimated at 127 parts by Andral and Gavarret; lower and higher figures have been given by other analysts, but this probably is the result of somewhat different modes of proceeding. In females the proportion of globules is lower, Becquerel and Rodier make the difference to be about 15 parts per 1000. The blood of the fœtus appears to contain an unusually large amount of globules, stated by Denis in the proportion of 222 to 140 of maternal; after birth this gradually diminishes. Plethora is the chief pathological condition in which an increase of red globules has been observed. One of the cases of cerebral congestion mentioned by Andral presented an amount of 138, 6 parts per 1000, an excess of 11 above the normal figure; after venesection, the globules in this case were so far diminished that the quantity only amounted to 101, 1 per 1000, considerably below the mean. In various febrile diseases, an augmentation of the globules has also been observed; thus, in the period immediately preceding the outbreak of continued fever, their amount was once found as high as 157.7; in the early period of a case of severe inflammatory fever, the fourth day of the disease, the globules had attained the extraordinary height of 185 parts per 1000, the greatest amount ever observed; in several cases of typhoid fever (fever with intestinal complication) the globules had risen to 142 or even 149, and

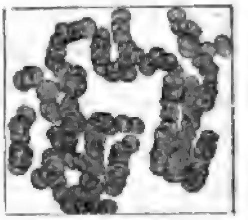
even on the second bleeding were found still considerably above the mean; in scarlatina and in measles an increase in the amount of globules was also found, the maximum (which existed in the latter) being as much as 146. No increase was observed in cases of variola or of modified smallpox. The condition of the general system coexisting with, and probably occasioned by, the increase in the amount of red globules is exaltation of the animal heat, heightened sensibility, and muscular irritability; the spirits are high, and the mental energy great, the pulse beats full and firm, the power of resistance to debilitating and morbid influences is considerable, the tendency in disease is to active inflammation and high febrile excitement, and bleeding, if employed, is well borne. Probably, a chief cause of danger when the amount of red globules is considerably increased, is the simultaneous diminution, or at least non-proportionate increase of the fibrin; hence arises a liability to serious hemorrhages in the brain or other parts. The effect of iron in promoting an increase of the red globules is well known, but it will often fail where the attendant circumstances are unfavorable, and we have seen a much more rapid effect produced by the change from a scanty to an ample diet. Free exposure to fresh air and light seems also powerfully to promote the formation of red blood, as much as the deprivation of them tends to destroy it. The opposite condition to plethora, for which the term spanemia (σπανος, scarce) is more appropriate than anemia, is essentially characterized by a deficiency in red globules. Extreme cases of this state are by no means unfrequent, and, which is not sufficiently known, are by no means unattended with serious danger. Sudden death has in several instances taken place apparently from the cessation of the heart's action, the debilitated organ being insufficiently stimulated by the impoverished blood. In an extreme case of chlorosis, Andral found the globules at so low an amount as 38.7 per 1000, the water at the same time being increased from 790 to 868.7. Similar alterations were found in the blood of other individuals who had become anemic from other causes, from lead poisoning, the cancerous or tuberculous cachexia. In Bright's disease, there is evidently a marked failure in the power of producing red globules, and the same is the case in the peculiar affection termed leucocythemia, which either depends on, or is coincident with, great hypertrophy of the spleen. We are inclined to think that in spanemic states the red globules are not only deficient in number, but defective in quality; they appear under the microscope manifestly paler than those of persons who have a healthy color, their hæmatin, in all probability, is not properly formed. The well-known symptoms of this condition are general debility, diminished temperature, palpitation, often excessive, of the heart, and various nervous affections. In some of the most malignant fevers the blood-globules appear to be actually destroyed; Dr. Williams has observed this in a case of albuminuria, proving fatal by purulent infection, and in a case of malignant scarlatina; we have examined the blood a few times in persons dying of such diseases, but have not found any noticeable alteration in the globules. Rokitansky mentions that he has observed in a septic state of the blood an altered condition of the globules, which are swollen up from their natural disk-like shape, and have parted with much of their

hæmatin to the surrounding fluid. The red globules, as has been mentioned, are very liable to be affected by the fluid in which they float; when this is dilute, they distend themselves by endosmosis, and become spherical, while the mass at the same time assumes a dark red color. By the addition, however, of concentrated saline solutions, the bright red color is again restored, and the corpuscles again assume their biconcave form. When carbonic acid is added to arterial blood, the corpuscles change their biconcave for the biconvex form,[1] and the color at the same time changes from red to black. On these facts, Scherer and Nasse maintain the theory that the alteration of color in the blood which is effected in the lungs, depends upon an alteration in the form of the corpuscles, the biconcave disks acting as concave mirrors, which collect the reflected rays instead of dispersing them as convex surfaces do. Whether this theory be true or not, the facts it records are certainly worth remembering, as a distended state of the corpuscles, by whatever cause occasioned, must greatly increase the liability to the occurrence of local congestions. When the fluid in which the corpuscles float is more aqueous than natural, the red fluid which they contain passes out of them by exosmosis, and mingles with the serum. When this is the case, the walls of the vessels, and the tissues in immediate contact with them, very commonly become saturated with a red color, which must be carefully distinguished from that which accompanies inflammation. The effect of diluting the Liquor Sanguinis upon the blood-globules is exceedingly well shown by an experiment performed by Mr. Lane. An animal was bled, and the serum, after the formation of the clot, was, as usual, colorless, or of a light yellow; a certain quantity of matter was then injected into the veins, and soon after blood was again drawn. The serum of this, however, was of a decided red, contrasting strongly with that of the preceding quantity. It was, therefore, quite clear, that the hæmatin had been removed from the globules, and dissolved in the serum. We have observed the same thing with the microscope in the blood of frogs, and in that of fœtal vertebrata. When the globules have not been exposed to the action of water, or only in a slight degree, the space between the envelop and the nucleus is filled with red fluid, which almost or entirely conceals the latter; but when water has been freely added, the red fluid entirely disappears, and the nucleus comes clearly into view. It seems very probable, as Dr. Williams suggests, that the instances of sudden death occurring immediately after copious draughts of cold water in an exhausted state of the system, have been in some measure owing to such alterations of the blood-globules, as we have just noticed. The same changes may also be concerned in the phenomena consequent on excessive losses of blood, especially in occasioning the local congestions which are apt to take place in them, and in the anemic generally. The red globules of human blood exhibit a tendency to cohere together in such a manner as to form tolerably regular piles, or rouleaux; in the healthy condition the cohesion soon ceases, and is not nearly so strongly manifested as in the inflammatory state. Of this, indeed, it is quite characteristic that the globules form

[1] The authors of the Physiological Anatomy, however, do not confirm this statement.

rows of some length, made up of numerous disks cohering together by their surfaces, and having their edges disposed so as to form a tolerably straight line. The cause of this tendency is not certainly ascertained, but the phenomenon seems to give some countenance to the opinion of Mulder, that the red corpuscles become invested in the lungs with a film of oxidized protein matter, which is found more abundantly in inflammation, and may cause the adhesion of the particles to each other. There must, however, be some further reason why the disks should so exactly adapt themselves to each other *face to face*.

Fig. 3.



Blood-corpuscles, magnified 400 diameters.

While so much uncertainty prevails respecting the origin of the red corpuscle, it is not possible to point to any foregoing condition as specially tending to promote their growth and increase, or to occasion their atrophy. All that can be said is, that a proper constitution of the Liquor Sanguinis is certainly essential, as being the material out of which these floating cells are nourished and built up. If this be impoverished, or otherwise deteriorated, the corpuscles will not be properly developed; and again, by improving the quality of their plasma, their healthy condition will be restored. Of this, we have a good example in those cases of chlorosis, where the administration of iron is sufficient to reproduce the ruddy hue of the complexion. In other cases, a defective state of nervous influence, proceeding from some mental affection, occasions the atrophy of the red particles; but we cannot tell whether this cause acts upon them primarily, or, as is more probable, through the medium of other organs and functions. There can be no doubt that the blood-globules have an appointed period of existence, after which they naturally decay. This decay probably takes place in the general course of the circulation, at least in part. Evidence, however, has recently been adduced to show, that the spleen is especially the seat of a destructive process, affecting the globules; and that the yellow pigment matter, so frequently found in this organ, is, in fact, the remains of their altered hæmatin. In the liver, also, the blood-globules seem to yield up their coloring matter, to furnish the yellow pigment of the bile; and the coloring matter of the urine is no doubt derived from the same source. The circumstance that so much pigmentary matter should, by these two channels, be ejected out of the system, shows, on the supposition that it is derived from that of the red globules, how rapidly the latter must undergo decay; and, by consequence, how fast their reproduction must take place. It is worth remarking, that one drug, the most commonly used, perhaps, of all, seems to have almost as much tendency to cause the destruction of red corpuscles, as iron has to promote their formation. This is mercury, under a course of which, as Dr. Watson mentions, a patient was blanched as white as a lily, who previously had a complexion compounded of the rose and the violet. The non-depuration of the blood mass exerts an injurious influence upon the development of the red globules, as well as upon its other constituents. The effect of some

diseases of the spleen and of the uterine system, which have been observed to be especially connected with an anemic state, may be explained on this principle. A quantity of blood congesting, and distending the venous channels of the spleen, is detained there longer than it normally should, and becomes more or less impaired in its composition and spoiled, so that when it again returns to the circulation, or any portion of it is conveyed thither, it acts after the manner of a poison upon the remaining blood mass, corrupting and contaminating it, and thus inducing general cachexia. Suppression of the menstrual discharge seems to act much in the same way, the blood not undergoing its periodical depuration, contains a quantity of effete matter, which reacts injuriously upon it, and, after a time, produces a manifest alteration from its healthy composition.

Of the morbid changes of the white corpuscles, we know indeed very little positively. Their number is, we think, increased in inflammation, but perhaps not to the degree that it has often been supposed to be. Mr. Wharton Jones remarks, that the quantity of white corpuscles existing in the blood naturally, has been estimated at less than it really is; and that, on the other hand, the accumulation of colorless corpuscles in the vessels of an inflamed part, and in the buffy layers of coagulated blood, has been referred to as proving the existence of a like quantity in the general mass of the blood. In blood from a pregnant female in the eighth month, the quantity of white corpuscles appeared to me to be increased, and their contents also less finely mottled, and more granular than in those of ordinary blood. In leucocythemia, the quantity of colorless corpuscles appears to be enormously increased, and their size exaggerated; but some doubt may be entertained whether these colorless corpuscles are of the same nature as those normally existing. In pyæmia, Lehmann says, that the colorless corpuscles are increased. Some of those observed, however, may have been of puriform nature.

There does not appear to be any positive instance of diminution of the colorless corpuscles.

Having discussed the pathological changes of the corpuscles or cells of the blood, we next come to consider those of the fluid in which they float. This fluid, the Liquor Sanguinis, consists of a watery solution of certain inorganic salts, in which there are besides dissolved a large proportion of organic substances. These are distinguished into fibrin, albumen, extractive matter, and oil. We shall consider each of these separately.

When fresh-drawn blood is left to itself, it very soon, as we know, passes from a liquid state into that of a solid mass; it coagulates. This change can be conclusively shown to be occasioned not by the corpuscular elements cohering together, as was once supposed, but by the solidification of one of the constituents of the fluid portion of the blood. Müller's experiment of filtering frog's blood, so as to separate the corpuscles, was decisive as to this point. The substance which thus spontaneously solidifies is termed fibrin, and the name evidently has reference to an inherent tendency which it possesses of assuming a fibrous arrangement. Fibrin, when separated from healthy blood, is

a tough, tolerably firm, elastic, stringy, whitish-gray-looking substance. It is insoluble in water, and sinks in this fluid, or even in the serum from which it has been separated. Under the microscope, it appears as an homogeneo-granular blastema, or basis-substance, with more or less marked tendency to fibrillate, or form actual fibres. The white corpuscles of the blood are very commonly seen imbedded in the fibrinous mass, but they do not appear to contribute to modify its character. This, at least, is true of healthy fibrin; of diseased we shall presently

Fig. 4.

Fibrils of Healthy Fibrin, entangling red and white blood-corpuscles (three of the latter are figured separately), and a few fibrinous fibrils.

speak. Lehmann describes as follows the actual process of coagulation of the fibrin as seen under the microscope. He says: "There appear here and there individual points of molecular granules, from out of which very soon extremely fine straight threads spring, which go off in a radiating manner from that point, but do not form star-shaped masses, as in crystallization; these threads elongate themselves gradually more and more, and cross with those which have proceeded from other solid points, so that at last the whole field of view appears, as it were, covered over with a fine, but somewhat felted network. Afterwards this network still proceeds to thicken, and the colorless corpuscles imbedded in it are often scarcely perceptible." With regard to its chemical composition, Lehmann remarks very properly, that fibrin, such as we obtain, cannot by any means be considered as a pure substance; it must contain the white corpuscles, and probably some amount of albumen and extractive matter. However, such as we find it, all the *chemical* difference between it and albumen appears to be, that there is a small quantity more oxygen in it than in albumen. This fact testifies strongly that fibrin is characterized much more by its vital endowments than by its peculiar composition, and would lead us to regard it as a modification of the albumen of the serum produced for some special purpose. This may be, in part, to give to the fluid portion of the blood a proper degree of spissitude, so that the corpuscles are better sustained and mixed with it, and that the circulating stream passes more readily through the capillary channels than it would do if it were more dilute. One very important end which the fibrin serves, is the formation of coagula at the orifice of wounded vessels, preventing the occurrence of further hemorrhage; and this is well illus-

trated by those instances occasionally met with of almost irrepressible hemorrhage from the slightest wound, in which it seems pretty certain that their blood must be very deficient in healthy fibrin. But a still higher dignity has been assigned to the fibrin; it has been regarded very generally as the plastic material, *par excellence*, from which all the tissues are formed, and the small amount of it present in blood was supposed to show that it was constantly being drawn off for the nutrition of the tissues as fast as it was formed. Of late years, however, much evidence has been adduced, which at least goes some way to establish an opposite view, and throws considerable doubt on the correctness of the old opinion. We will review the arguments on both sides.

In support of the specially plastic and organizable character of fibrin, it is argued that it seems to be by its means that the reparation of wounds is effected. A thin layer of coagulating, fibrillating material is the medium which unites and holds together the divided surfaces, and forms the first organic connection. So also when a fluid containing much fibrin is effused on the surface of serous membranes, it very commonly forms bands of adhesion passing between the opposed layers. These new-formed structures resemble very much normal, white, fibrous tissue. There is evidence to show that layers, and even masses of fibrin, are capable of being organized; so far, at least, as to become fibroid tissues, and that vascular networks are developed in them. Andral mentions a case of apoplectic effusion in the substance of the brain, in which—death having occurred many years after—a mass of pale red color, and fibrous appearance, and traversed by numerous small bloodvessels, anastomosing with those of the brain, was discovered. M. Louis has recorded the occurrence of a vascularized coagulum in a tuberculous cavity in the lungs; and many similar instances have been noticed, in which the fibrin of effused blood has evinced its capacity for organization. But how lowly is this organization; never in any known instance amounting to more than the formation of a fibrous tissue, more or less closely resembling the natural. This, almost of itself, is a proof that fibrin is not the peculiarly organizable and plastic element that it has been considered to be. It may also be said that albumen, which is the only other organizable constituent of the blood, shows no tendency, even when collected in large quantity, as in the fluid of ascites, to pass into any organized form; while fibrin, when effused, does at least assume the appearance of a lowly organized tissue. The condition, moreover, of the fibrin seems to be in some manner an indication of the vigor and health of the system. If it contracts well, and forms a firm, dense clot, there is reason so far to conclude that the constitution is sthenic and unbroken; but if, on the contrary, the clot be soft and easily broken up, the system is probably in an opposite condition. The above statements seem to amount to this, that fibrin is certainly capable of assuming a low type of organization; but they entirely fail to show that it is the special blood-constituent which is applied to the nutrition of the different tissues, and that therefore its abundance is a sign of vigor and health.

On the other side, the counter-evidence which we have to adduce is certainly of great force. Bleeding, which we saw to have a powerful

effect in diminishing the quantity of red corpuscles, has none such upon the fibrin; nay, it rather seemed to tend to increase it. In Andral's ninth case of articular rheumatism, the fibrin at the second bleeding was 7, and at the third 6, while in the first it was 5.4. In the tenth case, at the fourth bleeding, it presented the extraordinary figure of 10.2 per 1000, while in the first it did not amount to more than 6.1. The globules, however, were reduced by the three subsequent bleedings from 123.1 to 101.0. Starving also, instead of lessening, was found to increase the quantity of fibrin. An increase in this element was found in meagre, half-starved horses, amounting to as much as 7 or 8 beyond the healthy mean; and in one case, where no food was given for four days, the quantity of fibrin was found increased from 5 to 9. On the other hand, the improvement of a species which we found to be marked by an increase in the proportion of globules, seems to be also characterized, though less strongly, by a diminution in the quantity of fibrin. The average quantity of fibrin in a flock of sheep of pure blood was determined by Andral to be 3.1; in a cross-breed flock, the average was only 2.8. The blood of the fœtus and of the new-born animal, in whom certainly development and growth are proceeding rapidly, and in whom there must, therefore, be a constant demand for plastic material, is rich in globules, but poor in fibrin. Andral found in lambs, during the first twenty-four hours after birth, a proportion of fibrin only amounting to 1.9 per 1000; at the end of the fourth day, it had risen to 3.0 parts. This observation, as well as the familiar effect of diseases preventing the due oxygenation of the blood, in causing it to remain fluid after death, which seems to imply the non-formation or destruction of the fibrin, points very clearly, as it seems to us, to the conclusion that fibrin is an oxidation-product, and rather belonging to the descending series of destructive assimilation, than to the ascensive, plastic, and formative.

That fibrin takes an important part in the reparative process cannot be doubted; we constantly find it forming the uniting medium between divided parts, but have we any evidence that it becomes further developed, and passes into the form of any tissue more highly organized than that of the cicatrix? Surely, there is not the least; or rather, all that we know of the process of reparation tends to contradict such an idea. The case of a divided nerve serves to illustrate this point very well. Within a short time after the operation, the cut ends of the nerve are united together by fibrinous effusion, which has solidified round them; this passes afterwards on into the form of imperfect fibrous tissue, and so the apparent continuity of the nerve is restored. But we know that it is not really restored until very much later; we know that many months must elapse ere the severed nerve-tubules can be again connected by their own proper tissue, and their function restored. Now in this really reparative act there is no reason to believe that fibrin takes any prominent part, that it is at all more concerned than the albumen and oily matter of the blood, which are the elements chemically considered of which the nerve-matter actually consists. In the same way, in every organizing act in which there is more than the mere coagulation of

fibrin, albuminous serum is also present, and we have no reason to exclude it from participating in the formative process. Once more, if we take instances where deposition of fibrin has almost exclusively taken place, do we find development and growth proceeding actively in these deposits or not? The well of an aneurismal sac is often lined with dense layers of coagulated fibrin, and yet Professor Simon testifies that, on the most careful examination of these layers no trace whatever is to be found of new organization. So it is with the masses of fibrin that are deposited in the spleen, the kidney, and in other parts. They show, after a time, a tendency to retrograde and dissolve, but none to develop into any higher grade. The small, fibrinous, sub-pleural nodules often seen in tolerably healthy lungs, are favorably circumstanced as regards vascular supply, for growth and further development, but they do not appear to enlarge, or manifest any vital activity. The deposits of fibrin upon the valves of the heart, the wrongly-called vegetations, in like manner show no innate capacity of development and growth; they tend to contract and harden, or to become penetrated with calcareous matter, and never show any organized arrangement beyond a low grade of fibrousness.

The circumstance which seems to have contributed most, or at least very highly, to establish the opinion of the highly plastic quality of fibrin is, that its quantity is found to be so very largely increased in sthenic inflammatory diseases. Not only its appearance in the thick, buffy coat, but the quantity of solid effused matter, forming layers of false membrane, of considerable firmness and thickness, together with the character of the attendant symptoms, inspired an idea that the substance thus abounding in these diseases of marked sthenic type, and so-called increased action, and comparatively deficient in affections of asthenic type, was that which was especially plastic, and employed in the construction of all the various tissues in healthy nutrition. Dr. Williams says of fibrin, that it is a representative of the active state of the processes of reparation, circulation, innervation, and of those of nutrition and reparation, and that "it therefore exists in larger proportion and higher perfection in arterial than in venous blood." Dr. Carpenter, mentioning the solvent power which a solution of nitre has upon fibrin, remarks, that this only applies to venous fibrin, and that arterial fibrin is not thus soluble. As the solution of venous fibrin is coagulable by heat, it appears to resemble albumen, but the insoluble precipitate which it lets fall on exposure to the air, seems rather to possess the properties of arterial fibrin. "Hence," he adds, "it may be inferred, that the fibrin of venous blood most nearly resembles albumen, whilst that of arterial blood and of the buffy coat contains more oxygen, and is more highly animalized." We cannot agree with this inference, as an increased degree of oxidation is rather a sign of destruction than of development, at least as applied to the tissues. Moreover, the facts do not seem altogether established; Lehmann says that, in the human subject, whether it were taken from venous, or arterial, or inflammatory blood, he has found the fibrin soluble in nitre solution, excepting two cases of inflammatory blood. Pig's blood fibrin, whether arterial or venous, dissolved equally well and very quickly. This sup-

posed difference between arterial and venous fibrin is not constant. Thus much for the "higher perfection." As to the greater quantity which is found in arterial blood, it may be observed, (1.) That it is quite possible that it is employed to feed the various excretory glands, and is thus thrown off from the system, so that it exists in a less proportion in the returning venous current. (2.) That if arterial blood contains more fibrin, it contains also less albumen[1] than venous, so that, regarding fibrin as a product of oxidation, this is no more than might be expected; the fibrin being reconverted into albumen in the venous blood.

We shall conclude this discussion as to the nature of fibrin, with the following quotation from Rokitansky, whose testimony on this point cannot but be regarded as of considerable weight: "Considering the frequency of solid fibrinous blastemata as the basis of pathological new formations, compared to their rarity in the physiological condition, and referring also to the predominance of the development of the tissues out of cells in the physiological condition, and to the deficiency of fibrin in the embryo, we cannot suppress the declaration, that we are inclined to see (with Zimmermann) in fibrin really an excretory formation, a substance brought by oxidation nigh to the state of disintegration, an albuminous matter consumed by oxidation, which together with albumen appears only in the form of pseudo-fibrin to be any longer applied to nutrition." While thus rejecting the doctrine that fibrin is the sole or chief plastic element, we do not wish, on the other hand, to deny its importance when of proper quality in maintaining the due consistence of the blood, and in fulfilling such other purposes as those at which we have glanced. We now proceed to consider the variations of this substance, in different morbid conditions as to quantity and quality.

The average for the fibrin of ordinary venous blood, adopted by Andral, is 3 parts per 1000, but most observers place it rather lower. Scherer found in the blood of healthy men 2.03—2.63 parts. Its quantity as mentioned is greater in arterial than in venous blood, and is said by Schmid, after numerous examinations, to be only one-third in portal venous blood of its ordinary amount in that of the jugular vein. Sex does not appear to affect the proportion of fibrin, but the state of pregnancy does materially; in the first six months the quantity is decreased, the average being 2.3; during the last three months the quantity is increased, so as to average 4. After parturition, the quantity for a time seems to be still further increased; a circumstance which may have some relation to the tendency to uterine inflammation, and mammary abscess, which marks this period. In very early infancy, the quantity of fibrin in the blood appears to be small, but it experiences a marked increase at the period of puberty. Lehmann, in experiments upon himself, and Nasse, in experiments upon dogs, found that animal diet increased the proportion of fibrin above that which was found under a vegetable diet.

[1] Lehmann found in serum of the venous blood of a horse 11.428 parts of albumen per 100; in arterial blood 9.217 parts per 100. The quantity of fibrin in the venous blood (jugular vein) was 5.384, that in the arterial blood was 6.814.

Passing now to the morbid conditions in which the fibrin is found increased, we find, as a general law, that in all inflammatory diseases especially this is the case. In acute articular rheumatism, Andral records in one case as high a proportion of fibrin as 10.2 per 1000, in several others it amounted to 6, 7, 8, or 9. In bronchitis (acute), the highest figure obtained was 9.3, in pneumonia, 10.5. The maximum in pleurisy was 5.8, in peritonitis, 7.2. The increase was nearly the same in one case of erysipelas, in which it amounted to 7.3, and in another of tonsillitis, in which it was 7.2. A very important circumstance, well shown by Andral's tables, is, that the effect of bleeding was not to diminish the fibrin; in this respect, there was a marked contrast between the fibrin and the red globules; the latter sunk with each successive abstraction of blood, while the former most often rose, sometimes considerably. The following examples of this fact are very striking:—

	1st bleeding.	*2d.*	*3d.*	*4th.*	*5th.*	*6th.*
Acute Rheumatism	6.1	7.2	7.8	10.2	9	7
Pneumonia . .	7.1	8.2	9.0	10.0		
Peritonitis . .	3.8	4.7	6.1			
Pleurisy . .	3.9	5.8				

In tuberculization of the lungs the fibrin shows a decided increase, which, however, is most marked when intercurrent inflammation is set up in the part. In the crude state of the tubercles the mean of the fibrin is about 4; when softening has commenced, it is about 4.5; when cavities have formed, it is from 5 to 5.5. The red particles steadily decrease from the first. In chlorotic persons the quantity of fibrin is maintained at its usual average, and is sometimes a little above; this circumstance, taken together with the great diminution of the globules, accounts for the formation of a buffy layer on the surface, which is not uncommon in such blood.

Deficiency of fibrin is observed in very various morbid conditions. If we take as a certain indication the non-coagulated state of the blood (which perhaps, we are warranted in doing), it seems to be very deficient, generally, in all diseases proving fatal by asphyxia, or in which the respiration has been considerably interfered with for some time before death. Thus in cases of obstructive heart disease, the blood is very commonly in a fluid state, or very imperfectly coagulated, the coagula, such as they are, being very soft, and extremely friable. The same state is observed in cases of cyanosis, in which, owing to mixture of the venous and arterial blood, this fluid is never properly oxygenated. Excessive fatigue is said to prevent the blood from coagulating, but this has lately been contradicted by Mr. Gulliver, who found the blood coagulated in a hunted stag, and in two hares run down by harriers. Various poisons seem to have the effect of preventing coagulation of the blood; among these are hydrocyanic acid, carbonic acid, sulphuretted and carburetted hydrogen. Andral states, that if a concentrated solution of carbonate of soda be injected into a vein, the animal presents the symptoms of typhus, or scurvy (*i. e.* of a blood disease), and the blood is found fluid in the vessels. This statement is confirmed by Mr. Blake, and the same result proved to be produced by many other sub-

stances: others, again, though of very analogous character, produced a contrary effect. For instance, when caustic soda, or carbonate of soda, was employed, the blood was coagulated imperfectly, or not at all, but when liquor potassæ or its carbonate were used, the blood coagulated firmly. We have ourselves observed the formation of a buffy layer on the blood of an animal who had taken liquor potassæ for several days, to the extent of disordering its health. Nitrate of potash, and many other neutral salts, did not at all impede the coagulation of the blood, while arsenious and oxalic acid, infusion of digitalis, and some metallic astringent salts, did so decidedly. We may infer from these observations, that it is not the alkalies, as such, nor the neutral salts, as such, which produce the effects that are usually ascribed to them upon the blood, but certain substances of particular qualities. Again, in adynamic fevers, we often find the blood remarkably fluid, so as to gravitate after death to all the depending parts, and during life probably occasioning a tendency to hemorrhages, petechiæ, and vibices, which occasionally take place. The deficiency of fibrin in these cases is confirmed by analysis. Andral noticed some diminution of this element in the outset of continued fevers. It never increased, except on the supervention of an inflammation, and in the height of the disease sometimes sunk very low. In two cases, on the fifteenth day, it did not amount to more than 1. per 1000. A deficiency of fibrin might have been anticipated to exist in Purpura Hæmorrhagica; and, indeed, sometimes this seems to be the case. Simon, in his *Animal Chemistry*, gives two analyses; in one of which it is mentioned that there was no fibrin, in the other it only amounted to 0.905. Dr. Watson, speaking of this, or of the allied disease—scurvy—quotes a case, recorded by Huxham, in which "neither of the portions of blood that had been drawn, separated into serum and crassamentum as usual, though it had stood many hours, but continued, as it were, half-coagulated, and of a bluish-livid color on the top. It was most easily divided on the slightest touch, and seemed a purulent sanies rather than blood, with a kind of sooty powder at bottom." Dr. Copland and others refer to similar cases. On the other hand, Dr. Budd testifies that, in some cases of scurvy, the coagulation takes place as in healthy blood; and in two cases lately examined by Dr. Parkes, it appears that the fibrin, at least, was not in any great degree diminished.[1] Dr. Graves also mentions a case in which, after each of three bleedings, there was formed a firm coagulum, with a buffy coat. The plethoric condition, characterized as we have seen by an excess in the quantity of red globules, and evidencing a tendency to congestion and hemorrhage, is considered by Andral as generally associated with a deficient proportion of fibrin. He found in a strong, athletic man, who had symptoms of cerebral congestion, as low a figure of fibrin as 1.6; and remarks, that the minimum quantity occurred in those cases in which the symptoms of congestion were most

[1] In five analyses performed by Becquerel and Rodier, the general results were as follows: The clot was always of good consistence, the density of the serum low, the water increased, the quantity of globules diminished as well as their ferruginous contents; the fibrin was never diminished—sometimes increased; there was no increase of alkalinity or of salts; the solids of the serum were notably diminished.

intense. In a female, who had been struck down senseless by an attack of apoplexy, the first bleeding showed the small proportion of 1.9 of fibrin, the globules being at 175.5. After three days, when consciousness had begun to return, she was bled again; and now the fibrin amounted to 3.5, while the globules had diminished to 137.7.

Alterations in the *quality* of the fibrin manifest themselves very clearly in the varying size and firmness of the coagulum, which forms in blood drawn from a vein, as well as in the peculiarities of structure which microscopic examination reveals. We will consider the general and the textural differences separately. To estimate aright the condition of the coagulum of the blood, a full stream should be allowed to flow from a sufficient orifice into a deep vessel, which should be afterwards covered over, and should have been previously warmed. If drawn in a small, trickling stream, and received into a cold, shallow vessel, or if subsequently agitated, the coagulation is disturbed, and takes place either too rapidly, or forms, in the case of being agitated, irregular shreds.

The coagulum, when formed, may be very large and firm, so as to offer considerable resistance when an attempt is made to divide it. This implies a fair proportion, or, perhaps, rather increased, of healthy fibrin, with a considerable amount of red corpuscles. I have been informed of the case of a plethoric female, whom it was necessary to bleed frequently, during her pregnancy. The coagulum was described to me as so firm "that it might have been kicked from one end of a room to the other." On the other hand, the coagulum may be large, but so lax as to be very easily divided, and, if handled, readily breaking up. This implies either a deficient quantity of fibrin, or a defective contractile quality, or most commonly both. It may generally be taken as a positive sign that bleeding is not necessary, and that it will not be borne well. The coagulum, again, may be very firm, but considerably shrunken and contracted, manifesting this not only by its recession from the sides of the vessel, but by the concavity of its upper surface, which at the same time is covered with a layer, more or less thick, of a light-yellow color. This layer is fibrin separated from the red corpuscles, and is commonly termed the "buffy coat." In this case the quantity of fibrin is much increased, and probably also its contractile quality, which occasions the reduced size of the clot, and the drawing in of the surface, or, as it is called, "the cupping." A buffy coat sometimes forms on the surface, of clots which are rather deficient in firmness; it is, however, but thin, rather transparent, and produces no "cupping." It is mostly seen in rather asthenic conditions of the system, and from its appearance the blood which presents it is distinguished as "sizy." A very small, firm clot, with a more or less buffy surface, indicates a diminution of the red corpuscles, and at least a relative excess of fibrin.

The following circumstances[1] are favorable to the formation of the

[1] Dr. Todd and Mr. Bowman's "Physiological Anatomy," vol. ii. p. 295. It must be remarked, however, with regard to diminished density of the serum, that Mr. Gulliver has shown that in serum rendered thicker, heavier, and more viscid, by the addition of mucilage, the red corpuscles subsided not only more rapidly, but also more completely,

buffy coat: (1.) Slowness of coagulation, which gives the red corpuscles more time to sink. (2.) Increased weight of the corpuscles, and their aggregation together into rouleaux. (3.) A diminution in the specific gravity of the serum. (4.) A great diminution in the proportionate quantity of the red corpuscles, or an increase in that of fibrin. None of these circumstances, however, seem to us to account fully for the phenomenon in question; and we are much inclined to believe with Dr. Alison, that there exists an absolute tendency to separation between the fibrin and the corpuscles, somewhat, perhaps, of the nature of that which prevents the commixture of some dissimilar fluids. He remarks: "1st. That the formation of the buffy coat, though no doubt favored or rendered more complete by slow coagulation, is often observed in cases where the coagulation is more rapid than usual; and the coloring matter is usually observed to retire from the surface of the fluid in such cases before any coagulation has commenced. 2d. The separation of the fibrin from the coloring matter in such cases takes place in films of blood, so thin as not to admit of a stratum of the one being laid above the other. They separate from each other laterally, and the films acquire a speckled or mottled appearance, equally characteristic of the state of the blood with the buffy coat itself." It does not seem necessary to assume that there is any actual repulsion of the red particles from the fibrin, the tendency to separation may simply depend on the increased attraction (which is manifest) of the respective parts of each element together.

The differences in *textural quality* which fibrin often presents, have been admirably described by Rokitansky and Mr. Paget. We will follow the account given by the former, though it be somewhat in minute detail, as we may have occasion hereafter to refer to it. Taking strongly marked typical examples, he describes, 1st, the fibrin which is found in the bodies of healthy persons. This forms pretty compact and tough, moderately adhesive coagula of a whitish color, passing into yellow. They may be torn into membranous layers, and exhibit along the torn surface a delicate felting. Microscopic examination shows a clear (hyaline) membranous or flaky basis-substance, and upon it a thick felt of very fine, very elastic, black-bordered, branching fibres, quickly dissolved by acetic acid, which project out free at the borders of the specimen. Together with this there are seen on the preparation, appearing more evident after treatment with acetic acid, round, glistening nuclei, and along with these a few delicately-granulated, dull-looking, round, and oval nuclei, and similar cells of the size of a pus-corpuscle. Of the same composition are certain soft, brawn-like coagula, termed *Pseudo-fibrin*, which Rokitansky regards as a very important transition-formation as regards healthy nutrition, between albumen and fibrin.

The 2d variety of fibrin forms coagula much like those of the preceding, but of more adhesive quality. They include often in their substance

than in serum, which was rendered thinner, lighter, and less viscid, by being mixed with a saline solution. Mr. Wharton Jones—from whom we have, in part, taken the above passage—believes that the viscid state of the liquori sanguinis promotes the subsidence of the red corpuscles, by increasing their natural tendency to aggregate together.

notable quantities of serum. Under the microscope they are seen to consist of a flaky basis substance, which divides itself into fibres, either flat, or roundish, harsh, and stiff, or resembling those of organic muscle; or the basis-substance may appear sometimes membranous, most delicately fibred, with a wavy crisping. On this basis there occur, together with elementary granules, numerous round black-bordered, sometimes also staff-shaped or fibre-like, elongated nuclei; and besides these, especially in the fluid which trickles out, there are granulated, dull-looking nuclei, and similar nucleated cells. This fibrin, with traces of the preceding, is often combined with the other varieties. Rokitansky says of it, that it is often effused, as the result of morbid processes and of inflammation, and that it must be regarded as diseased, but to be distinguished from the following varieties by the appellation of plastic or organizable.

The coagula of the 3d kind of fibrin are distinguished by their opacity, by a dull-white color, verging on yellowish, or yellowish-green. They often contain, besides serum, red corpuscles also in considerable quantity, which indicate an increased capacity of coagulation, and more rapid occurrence of the same. They present in this case different shades of redness and opacity. Microscopically examined, the coagulum is found to consist of a flaky or fibro-flaky basis mass, or of a dull, streaky membrane, each of which is rendered opaque by a great quantity of fine punctiform matter. Besides, there exist upon it, and together with it, in the serum, a great number of nuclear formations, and developed dull granulated nuclei, and similar more or less developed cells. Often, the whole coagulum appears to consist of both the last-named elements, together with some punctiform substance. The nuclear formations in general show the ordinary indifference towards acetic acid. This fibrin has less adhesive quality.

Fig. 5.

Corpuscular unhealthy fibrin, from exudation on pericardium. It consists of an homogeneous granular basis, imbedding numerous corpuscles.

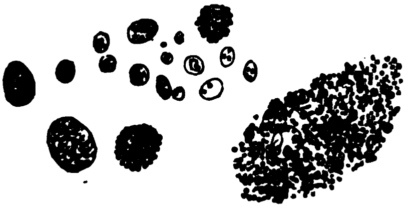
Fibrin of the 4th kind presents a still higher degree of the morbid condition of the preceding. The coagula are in the highest degree opaque. When they contain no blood-globules they approach more manifestly to a greenish-yellow. Often, however, they contain large quantities of blood-globules, and are reddish-gray, or reddish-brown, which indicates rapid coagulation. More closely examined, they consist of a thick, finely-punctuated mass, of nucleus and cell-formations, comporting themselves in various degrees more and more like the pus-cell and pus-nucleus, which are held together generally by a tenacious, amorphous, uniting mass; no network of fibres exists, and no other fibre-formation. It has lost still more of the adhesive (soldering) quality. The 3d and 4th forms Rokitansky denominates croupous.

At this point the fibrin approaches close upon that existing in Pyæmia; it has the croupous constitution; the nuclei and cells inclosed in the coagulum are true pus-nuclei and pus-cells.

Though the above quotation is long, yet the great importance of the subject, and the value of the description given by so high an authority,

made us unwilling to omit or abbreviate it. Professor Paget, agreeing closely with Rokitansky, expresses the same facts more shortly and simply. His description refers, however, to fibrin, as it appears in exudations, while that of the German pathologist is expressly confined to the fibrinous coagula which are found within the vascular system. The

Fig. 6.



Softening fibrin from a vein clot. The dark points are minute oil drops.

correspondence, however, between the fibrin *in* and *out* of the vessels is so close, that the characters of the one apply also to the other; and we may, therefore, say, that all the varieties of the fibrin of the blood, as manifested by the condition of the coagula, depend upon the predominance of one of two forms of structure. In healthy fibrin the fibrous structure greatly predominates, the whole mass fibrillates more or less perfectly, and the included corpuscles are comparatively few. This is Mr. Paget's *fibrinous* variety. Unhealthy fibrin, which tends to disintegration, consists of a granular mass, imbedding very numerous nuclei and cell-formations; this is the *corpuscular* variety. These two correspond to Rokitansky's 1st and 4th varieties, and his 2d and 3d are only combinations of them in different proportions.

We shall here briefly notice the metamorphoses which coagula of fibrin may undergo: (1.) A more or less perfect development of fibre may take place, in which the nuclei are chiefly concerned. (2.) The coagula may fall to pieces, and undergo a kind of dissolution into a pappy or pus-like fluid, or in some cases into a fluid which is really of the nature of pus. This change may befall coagula of healthy fibrin, in consequence of their being placed in conditions unfavorable to fibre-development, but is more particularly observed in the corpuscular varieties. It was shown by Mr. Gulliver, that the puriform fluid often found in vessels was really fibrin which had undergone softening; and it was an important step to prove that such collections of pus-like matter were not the result of phlebitis; but in recognizing their non-inflammatory origin, perhaps, it has not been seen clearly how very similar, or even identical, they might be with certain forms of true pus. (3.) The fibrin may part with some of its natural moisture, and change into a resisting, stiff, dull, translucent, or also opaque, horny mass. In process of time it may become ossified (probably calcified). (4.) It may undergo fatty transformation, becoming converted into a mass of small, oily molecules or drops. (5.) It may gradually be dissolved and taken up again into the circulation. Rokitansky mentions as an instance of this the removal of fibrinous vegetations from the cardiac valves.

ORGANIC CONSTITUENTS OF THE SERUM.

Albumen is the principal organic constituent of the serum, in which it exists dissolved in water in the proportion of 63 to 72 parts per 1000 of blood. The specific gravity of the serum is on an average 1028, and varies less than that of the entire blood. The serum is naturally of a light yellow color, which does not appear to depend on the presence of hæmatine or of bile pigment, but to be special to this fluid. We have already expressed our dissent from the doctrine that the fibrin is the sole or chief material intended for the growth and nutrition of the tissues, and fully believe that the albumen is quite as much or more applied to this purpose. We have no means at present of ascertaining numerous qualitative variations which probably affect the albumen of the serum. Indeed, we cannot but believe that its composition must be liable to continual minute changes, as on the one hand nutritive material for various tissues is drawn off from it, and on the other, chyle, scarce yet raised to the blood-standard, and lymph, containing effete or semi-effete residua of nutrition, are poured into it. All such, however, to a certain extent, are clearly natural; and were our powers of analysis greatly more refined, we should probably find that it was by the most gradual steps that physiological variations passed into morbid.

The amount of albumen, according to Andral and Gavarret, is notably increased in various diseases; but this excess does not appear to be characteristic of any. In acute rheumatism, an increase was found varying from 4 to 24; in pneumonia, the highest increase was about 12; in pleurisy, the extraordinary amount of 34 in excess was once observed; and several other instances are mentioned of lower degree. Peritonitis, tonsillitis, and erysipelas, all furnish cases in which there was more or less considerable increase of the albumen. This is the case also in tubercular disease of the lungs, and in simple and continued fevers. Bleeding does not appear to influence the quantity of albumen in a very constant way; on the whole it tends to decrease it, and this seems to be especially the case in typhoid fever. In cases of cerebral congestion and of apoplexy, generally considered as examples of plethora, there was found, especially in the former, a very considerable increase in many cases of the albumen. This amounted once to 24.8 beyond the mean. In the latter there were several instances of marked diminution. Cases of chlorosis, in which the globules are so remarkably diminished, show rarely any diminution of albumen; and sometimes a considerable excess, amounting to 14 or 20 parts per 1000.

Diminution of the albumen of the serum probably takes place in various diseases, attended with defective nutrition and wasting, but has been more particularly observed in renal dropsy. It appears as if the albumen of the serum, being drained off in the urine, and in the dropsical efflux, there remains behind a less quantity in the blood. This idea is confirmed by the results of three successive analyses of Andral's, in the first of which there being much albumen in the urine, the serum contained only 57.9; in the second, the urine containing less albumen, the amount in the serum had increased to 66; in the third, the urine being no longer

albuminous, the quantity in the serum had returned to its normal figure, 72. It is also in accordance with the circumstance noticed by Dr. Bright, and others, of low specific gravity of the serum in this disease. Still, this explanation does not accord with the general fact that dropsical effusions are more watery, and contain less albumen than the serum, which one would therefore expect to find of greater density in such cases. Probably the supply of such albumen, both from the chyle and lymph, is defective in quality and quantity. Becquerel and Rodier state, that in diseases of the heart the albumen of the serum varies but little as long as there is no dropsy; then it diminishes, and often considerably. In the disease called the *rot*, affecting sheep, which is characterized by the presences of numerous distomata (flukes) in the biliary ducts, M. Andral and his coadjutors found the albumen of the blood considerably diminished, as well as the red globules, while the water was greatly increased. Sheep are also subject to ordinary anæmia, *i. e.* to a deficiency of red globules only in the blood, the albumen remaining at its normal amount; and it is very worthy of remark, that in these latter cases dropsy does not take place, while in sheep affected with the rot it is not uncommon. This seems to point out that when in cases of cachexia and debility serous infiltrations of the limbs occur, it is owing to a diminution in the quantity of albumen in the blood.

EXTRACTIVE MATTERS.

Chemistry has as yet ascertained too little respecting these substances, even in the healthy condition, to make any conclusion possible regarding their variations in disease. Simon's division into water extract, proof spirit extract, and alcohol extract, is of no avail for physiology or pathology. As his eminent namesake remarks, what we want is a division of these matters according to the organs or systems of organs that produce them. Still, the recognition of the existence of such matters in the blood is important, as showing us the actual presence of principles that are effete, or tending to become so in this fluid, and reminding us how often ill health and *malaise* may depend on the formation of unnatural products of this kind, which come at length to be generated by an almost habitual vice of the system.

According to Lehmann's estimate, the quantity of extractive matter in healthy blood is 0.25 to 0.42 per 100. Nasse found a larger proportion in the blood of children and of young animals than in that of adults. Arterial blood, according to Lehmann, contains more than venous blood in the proportion of 5.374 parts to 3.617. Portal vein blood of horses, five to ten hours after food, contained on an average 7.422 parts; twenty-four hours after food the quantity amounted to 10; but it was always less than that existing in hepatic vein blood, which averaged above 18. The existence of a larger quantity of extractive matter in arterial than in venous blood, may perhaps be accounted for by the increased oxidation of some of the organic matters which takes place in and after the passage of the blood through the lungs. The larger amount in the blood of young growing creatures is in corre-

spondence with the greater activity of their circulation, and their nutritive processes generally. The excess in the hepatic vein above the portal blood indicates that an absorption of matter from the hepatic cells into the current traversing the lobules takes place. Sugar, we know, is conveyed into the blood from this source, and there is equal reason to believe that extractive matter may be. Though we cannot class liver-sugar, or glucose, altogether with extractive matter, as it is rather of the nature of a secretion, yet we cannot but notice the probable relation in which it stands to the disease called diabetes, and the ideas which this view suggests as to various diseases which appear to have their seat in the blood. It seems that one very important feature in diabetes is, that the sugar formed in the liver, and absorbed into the blood, is not decomposed, as it ought to be, into carbonic acid, but circulating in the blood, and arriving at the kidneys, stimulates them unnaturally, and so occasions the profuse diuresis which exhausts the system. So, various ill-defined extractive matters, intended to be decomposed and eliminated through the skin, or other channels, may fail to undergo their normal changes, and be, in consequence, more or less completely retained in the blood, which, circulating thus contaminated, becomes a source of mal-nutrition and disorder to various parts. What more probable account can be given of the origin of most skin diseases? Surely, in such an instance as this the value of a sound pathology is most manifest, if it be only to open our eyes to the absurdity of not a few of the ordinary remedial measures. Liebig has particularly described three substances which appear to belong to the class of extractive matter, creatin, creatinin, and inosinic acid; the two former seem to be of the nature of alkaloids, the latter combines with bases as an acid. They are all nitrogenized substances, and are found in the watery extract of muscle; the two former are present in the urine, and are, therefore, doubtless effete. They do not seem to have been detected in the blood, but must of course have passed through it before being excreted with the urine. Creatin, Lehmann states, is analogous to Thein, an alkaloid, which in some trials has produced very severe nervous symptoms, even when taken in small doses. Many slight changes in creatin or the allied substances render them capable of producing any similar phenomena.

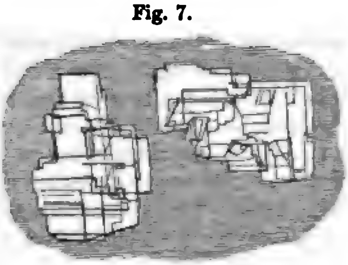
OILY MATTER IN THE BLOOD.

The quantity of oil existing in the blood cannot be estimated only from the amount contained in the serum, for it is present also in the red corpuscles, and in the fibrin. The quantity contained in the crassamentum, which is made up of these two components, is not much inferior to that in the serum. The serum of arterial blood contains less oily matter than that of venous. Lehmann gives 0.264 per cent. as the proportion in the first, 0.393 per cent. in the latter.[1] Chevreul

[1] Lehmann found in 100 parts of dried blood-corpuscles of the ox, 2.249 of oily matter: arterial blood-cprpuscles contained 1.824 parts of oily matter per 100 thereof; venous blood 3.595 parts.

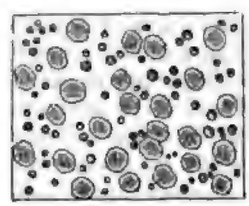
states the quantity of oily matter in fibrin as amounting to 4 or 4½ per cent. Lecame distinguishes a crystallizable and non-crystallizable oily matter in the blood, the former in the proportion of 1.20 to 2.10, the latter in that of 1 to 1.30 per 1000 parts of serum. Cholesterin and serolin are two crystalline fatty substances which have been found in blood; the former is of very common occurrence in exudations in various parts, and in some tumors; it forms the well-known rhomboid tablets, by which, when in a solid form, it is immediately recognized. The blood of females, according to Becquerel, contains on an average more fatty matter than that of men. In both sexes, the quantity of cholesterin increases with advancing years, after the age of 40 or 50. It seems established, as was natural to expect, that the quantity of oily matter in the blood increases after taking food. A milky state of the serum had often been observed, but though it was generally supposed to depend on the admixture of chyle, this could hardly be said to have been proved until lately. Dr. Buchanan's experiments upon healthy persons show that the serum "becomes turbid about half an hour after taking food, the discoloration increases during several hours, attains its maximum in about six or eight (after a full meal), and then becomes gradually clearer till its limpidity is restored. The opaque serum is generally milk-white, sometimes cream-yellow, or yellowish brown, like thin oatmeal gruel; or it merely loses its limpidity, and is like weak syrup. It always contains solid white granules, smaller than the blood-corpuscles, which are suspended in it, and which will rise in a white

Fig. 7.



Cholesterin.

Fig. 8.



Fat in Blood.

cream to the surface, either spontaneously, or after the fluid has been saturated with common salt. The cream thus obtained is soluble in caustic potash, but insoluble in ether or alcohol, and is considered by Dr. R. D. Thomson as probably a protein compound." The microscopic appearance of milky serum, as I have observed it, has depended on the presence of a diffused finely divided matter, much resembling the molecular base of the chyle. Sometimes, however, distinct oil drops are observed. When we remember that the molecular opaque matter of the chyle is surely of oily nature, and find that the opacity of the serum, which occurs after a meal of food containing fat, is in part due to the presence of protein granules, we can hardly help entertaining the idea which various other circumstances confirm, that the oily matter is actually converted into albumen. Should this ever be

proved to be the case, we should clearly see how in a lowered state of the vital powers, nutrition must be impaired, and how the failure of this, the first of the ascending nutritive processes, would be a prime cause of the degeneration of various tissues, or of the accumulation of adipose tissue, which are both signs of feeble general power. The general result of Becquerel and Rodier's very careful analysis of the blood in various diseases, with regard to the variations in the quantity of oil, is that almost from the outset of every acute disease the amount is increased, and particularly that of the cholesterin. Diseases of the liver, Bright's disease, and tuberculosis have the same effect.

SALINE INGREDIENTS.

There remain for our consideration the various salts of the blood, and the water which holds them in solution. The amount of salts in the blood of man is somewhat greater than in the blood of woman; that of the former contains, on an average, 8.8 per cent., that of the latter 8.1; in both sexes the variations compatible with health are considerable. The blood of adults contains more salts than that of children; arterial blood more than venous. The prolonged use of aliments containing much common salt is said to cause an increase in the proportion of the latter, and of the other salts generally. The following appear to be the principal saline combinations in the blood: Chloride of Sodium, and Potassium, Sulphate of Potash, Carbonate and Phosphate of Soda. Of the alterations which the salts undergo in different diseases we have not much knowledge. In malignant cholera, the excessive drain tells most on the fluid part of the blood, and hence that remaining in the vessels is thick and tarlike; hence, also, the extraordinary, though temporary, effect of injecting saline solutions, which return to the blood the material effused from it, and revive all the functions that were wellnigh extinct. Doubtless, if the intestinal discharges could be arrested, the effect would be permanent, but as it is, their effect is soon exhausted. Henle assumes that in inflammation, when the flow of blood in a part is retarded, exudation of the more watery and saline parts of the liquor sanguinis takes place, so that that which remains in the vessels becomes inspissated, and producing endosmotic changes in the red corpuscles disposes them to adhere together. This view, however, is merely hypothetical. Vogel states that "the salts are increased in scurvy, and it is very probable that this change influences the condition of the fibrin, hindering its coagulability, and, perhaps, checking its formation; that it affects the blood-corpuscles by withdrawing their water, rendering them granular, and collecting them in heaps; and that it thus plays an essential part in the disease itself." However, his statement has been contradicted rather than confirmed. In the cases of purpura examined by Dr. Parkes, which have been alluded to, the quantity of salts seems to have been below the average. In acute exanthemata, in dysenteric affections, in endemic agues, Lehmann mentions the proportion of the salts to be increased, as also in Bright's disease, in typhus, and in all kinds of dropsy and hydræmia. In violent inflammation the salts are much diminished.

WATER.

Lecame's estimate of the mean quantity of water in 1000 parts of blood is 790; he found more water in the blood of women than in that of men, more in the blood of children and of aged and debilitated persons than in that of vigorous adults, more in the lymphatic than in the sanguineous temperament. It is clear that the whole quantity of water is not contained in the serum, a certain proportion, which must vary with the specific gravity of the blood, is inclosed in the red corpuscles, and holds their coloring matter in solution. The serum of arterial blood and of portal vein blood is said by Lehmann to contain more water than that of venous blood generally. It is a very remarkable circumstance, and strongly indicative of wise provision, that it is very difficult to demonstrate by analysis an actual increase of the quantity of water in the blood after copious drinks have been taken. Denis and Schultz are at issue as to whether such an increase is detectable or not. This seems to show how exactly the vascular system is kept at a certain degree of tension, so that in proportion as absorption at one part takes place, excretion at another ensues correspondingly. The effect of bleeding and starving which was before noticed, of reducing the amount of globules, tells, of course, proportionally, in increasing the quantity of water; this can easily be ascertained, as it is in great measure relative; the merely positive increase is much more doubtful. Andral mentions a case of confirmed chlorosis, in which the water in the blood amounted to 867.9, an increase of nearly 78 parts per 1000. Lehmann states, that in the beginning of most diseases, especially acute ones, the blood is found more watery than natural, the serum, however, at the same time being richer in solid contents. He accounts for this, by supposing that the material which should have been applied to the formation of the globules, or which results from their decay, remains in the serum. During the first ten days of typhus, the first stage of scarlet fever, measles, and cholera, this increase in the watery constituents of the blood does not appear to take place.

The condition which is commonly called Anæmia should, it would seem, more properly be named Hydræmia, as in most cases it is not so much a deficient quantity of blood which it is intended to describe as a defective quality. Doubtless, a person reduced greatly by phthisis, or any exhausting disease, is really in a state of anæmia; he has less blood than natural in his body, as well as too watery; but a female suffering from consequences of amenorrhœa, with pale lips and face, is much more likely to be in a state of hydræmia, the mass of blood not being diminished, but its red corpuscles replaced by water.

Having examined the variations which the several constituent elements of the blood are liable to undergo, we next proceed to make a few remarks on certain abnormal matters which are occasionally present in it. *Carbonic acid gas*, the product of respiration, *i. e.* of the conveyance of oxygen throughout every part of the frame, becomes, if it accumulates beyond a certain small amount in the blood, the cause of serious disorder and speedily of death. Various diseases of the thoracic viscera, or impedi-

ments to the free action of the walls of the chest, prevent more or less the due oxygenation of the blood, which is indicated by the dusky hue of the complexion, the lividity of the lips, the sensation of oppression at the chest, and of dyspnœa. The larger the quantity of blood which is circulating in the vessels, and the more vigorous the state of health and the general activity of the functions, the greater must be the accumulation of carbonic acid when any asphyxiating cause begins to operate, and the more severely will its effects be manifested. If, however, the mass of blood be greatly diminished by exhausting drains, by diminution of food, and by non-development of its corpuscles, then the amount of respiratory action may be also greatly diminished without producing the symptoms above mentioned. Thus, if a person in health should suddenly be deprived of one-half or three-fourths of his breathing apparatus, he would quickly die, suffocated; but a patient in the last stage of phthisis, whose lungs are destroyed to the same extent, may continue to live on without experiencing any notable dyspnœal distress. In the same way, when, during violent exertion, a greatly increased quantity of carbonic acid is formed, the amount of oxygen introduced into the lungs requires to be increased in proportion, and hence the hurried and panting respiration. A hybernating animal scarcely breathes at all; its animal heat is not above the temperature of the atmosphere, and all its functions are in abeyance; carbonic acid, therefore, is not formed, and the inhalation of oxygen is not necessary; life, reduced to this low ebb, continues in an atmosphere which would cause instant suffocation if the animal were awake. Man does not hybernate, but it seems highly probable that the system must temporarily have been in a similar state in those cases in which life has been restored after prolonged immersion for half an hour or more. In persons affected with the *morbus cœruleus*, when, from some malformation, the pulmonic and systematic circulations are no longer kept distinct, we have the best opportunities for observing the effect of an unnaturally venous condition of the blood. The following excellent description is given by Dr. Williams: "Individuals thus affected are in a lower scale of animation. The slower processes of nutrition and secretion seem to go on pretty well, but the muscular power is low, slight exertions bring on symptoms of faintness, palpitation, suffocation, or insensibility, the animal heat is lower than natural, and there is greater suffering from the influence of cold. In short, all the powers of body and mind are slender, and are easily disordered by any circumstances which tax their activity. In the few that reach mature age there is no sexual passion. . . . . The subjects of cyanosis are said to be very liable to hemorrhages, and when these occur spontaneously, or from accidental causes, it is very difficult to stop them. This must be ascribed to the deficiency of fibrin, which we have already found to occur where the changes of the blood by respiration are imperfect."

In what way does accumulation of carbonic acid in the blood prove fatal to life? We find, after death from asphyxia, the left side of the heart comparatively empty, and its cavities contracted, the right side gorged with blood, as well as the veins generally; the lungs are also distended and gorged with dark blood. Now, it has been shown that

the essential cause of the failure of the circulation is not paralysis of the heart, or of the brain, though these may have some influence, but arrest of the blood in the capillaries of the lungs.

How this arrest is produced we have not sufficient positive information to enable us certainly to explain, but we see that it is a phenomenon of the same class as that congestion which has been mentioned as often occurring when the function of a part is suddenly put a stop to, or, as Dr. Carpenter expresses it generally, "the performance of the normal reaction between the blood and the surrounding medium (whether this be air, water, or solid organized tissue) is a condition necessary to the regular movement of the blood through the extreme vessels." The correctness of this position is almost demonstrated by the following experiment of Dr. Reid's: Having adapted an hæmadynamometer to a systemic artery of an animal, and obstructed its respiration, he found that when non-oxygenated blood was beginning to circulate, as shown by the commencing supervention of insensibility, the column of mercury in the tube was raised, indicating, of course, an increased resistance to the onward flow through the capillaries.

In this instance the normal changes between the non-arterialized and the tissues could not take place, and, consequently, the blood could not freely pass through them. The converse of this experiment is presented to us in the effect of extreme cold on parts that are exposed to it. The functions of the part are abolished, the circulation languishes and at last ceases, the vessels remaining congested with venous blood, which is not carried onwards. Hence the blue, or livid color which the surface presents. In this case, the vital power of the tissues seems to be paralyzed by the sedative influence of the cold, and as a consequence, their nutrition and circulation are also brought to a stand. The arrest of the pulmonary circulation in asphyxia seems very analogous to the foregoing instance; the normal changes in the lung tissue having ceased, the blood is no longer able to traverse its capillary plexus freely, but stagnates there, and congests the part. Thus far we simply class together a number of similar phenomena, and educe from the circumstances common to them a kind of law, viz: that quoted from Dr. Carpenter. But lately, Dr. Draper has brought forward a view which is extremely plausible and beautiful, and appears to us likely to prove of the greatest value in physiology and pathology. It is founded on the statement, "that if two liquids communicate with one another in a capillary tube, or in a porous or parenchymatous structure, and have for that tube or structure different chemical affinities, movement will ensue, that liquid which has the most energetic affinity will move with the greatest velocity, and may even drive the other liquid entirely before it." The essential idea appears to be this, that the on-coming liquid is attracted, particle by particle, to various points of the tissue which it traverses, that the attraction, having taken place, soon ceases, in consequence of an alteration being effected in the attracted fluid, and that then the particles of fluid, no longer retained or drawn to the part by attraction, are pushed on by fresh quantities of unaltered fluid, for which the tissue has attraction. Thus, in the systemic capillaries, the arterialized blood is attracted to the tissues, changed by the act of nutrition to venous, therewith loses

its capacity of being attracted, and is driven on by more arterial blood, coming up within the range of the tissue's attraction. In the same way we may conceive an attraction to subsist between the venous blood and the air in the cells of the lung, which will, of course, cease as soon as the change from venous to arterial blood has been accomplished. The effect of this nutrition force, a term which we prefer to Dr. Carpenter's "capillary force," is, evidently, to promote remarkably the free transit of the blood through a part, and there can be no doubt that the arrest or abolition of this force must tend materially to obstruct the circulation. Hence, in asphyxia, the attraction probably continuing some time, but the normal changes which liberate each particle from it not taking place, the blood continually arrives and stagnates in the pulmonary capillaries. We have dwelt the longer on this subject, because it appears to us of so much importance to recognize the principle that the nutrition of a part influences so materially the circulation of blood through it, and because we may often have occasion to refer to the view here enunciated. The poisonous influence of carbonic acid is well shown by the following comparative experiment of Rolando. He tied one of the bronchi in a tortoise, and found that the animal was not materially injured thereby; but when, instead of merely cutting off the access of air, he furnished a supply of carbonic acid to that lung, the other still receiving air, the animal died in a few hours.

When the action of the *kidneys* is arrested, or seriously interfered with in any way, their secretion products are no longer carried out of the system, but remain in and contaminate the blood. The effects produced by the blood thus poisoned are somewhat different, according as the secretion is more or less suddenly and completely suppressed. When the suppression occurs suddenly, the acute form of uræmia, as it is called, manifests itself. Frerichs describes three varieties of this. In the first, after some pain of the head, giddiness, or vomiting, the patient soon sinks into deep stupor, from which in no long while he cannot be aroused. In the second, epileptic convulsions suddenly appear, affecting the whole muscular system, and returning after occasional intermissions. The consciousness may remain unaffected. In the third form, both convulsions and coma occur. Such cases constitute the disease which received a separate name, as *ischuria renalis*, but they probably belong to the same class as the acute anasarca, which occurs sometimes spontaneously, or after scarlet or typhus fever, the anatomical characteristic of which is great sanguine engorgement of the kidney. Uræmia, in its chronic form, appears at the close of Bright's disease very frequently. Frerichs describes it as coming on gradually and unperceived, occasioning dull headache, or confused sensation, impairing the mental and bodily faculties, and producing some dulness and drowsiness. These symptoms may remit if the urinary secretion increases, or they may progress, and become more intense, the drowsiness deepening into stupor and coma. Vomiting is a frequent symptom in uræmia, and sometimes amaurosis, or disturbance of the hearing, is observed. Diarrhœa sometimes takes place, and seems to avert the dangerous consequences of uræmia; it was a prominent symptom in the animals whose kidneys were extirpated by Prevost and Dumas, and in those similarly treated

by Bernard and Barreswill; in the latter, it was particularly observed that large quantities of ammoniacal fluid were poured out by the mucous membrane of the stomach and intestinal canal; while these continued, the cerebral functions were impaired, but as soon as they ceased, the symptoms of intoxication commenced. Inflammations of the serous membranes, especially the pleura and pericardium, are very commonly produced by uræmic poisoning in a less severe form. Cases of pericarditis of renal origin are nearly as frequent as those of rheumatic. The term uræmia seems to imply that the poisoning of the blood depends on the presence of urea, and such has long been the general belief, but numerous experiments and observations of late have done much to invalidate it. The quantity of urea in the blood, and the intensity of the symptoms, bear no proportion to each other; there may be much urea in the blood and no symptoms, and severe symptoms with little or no urea in the blood. Dr. Rees, observing this, concluded that a watery state of the blood was the cause of the symptoms. Dr. Todd, Vauquelin, and others, have injected urea into the veins of animals, or given it by the mouth, without producing any other effect than increasing very greatly the flow of urine. Still more, Frerichs has repeatedly injected human urine into the blood of animals without producing any ill effects. It seems clear then that it is not urea, nor any other constituents of the urine, that produce, by their presence in the blood, the symptoms of poisoning. It may be, however, some of their decomposition-products; and Frerichs states that he has proved it to be the carbonate of ammonia, which is well known to result from altered urea. He has repeatedly demonstrated the presence of ammonia in the air expired by the sick, and by animals into whose veins urea had been injected after extirpation of the kidneys. Carbonate of ammonia, he says, can always be detected in the blood whenever uræmic symptoms exist, as well as usually traces of undestroyed urea. The two following experiments certainly go far to establish Frerich's theory of uræmic intoxication. When a solution of urea is injected into the veins of animals from whom the kidneys have been removed, no symptoms take place for some time; but after one hour and a quarter to eight hours vomiting commences, and convulsions, or sopor, and coma begin to appear at the same time that ammonia can be detected in the air expired. After death, ammonia in large quantity was found in the blood. "The brain and its membranes were normal in appearance, and the quantity of fluid in the ventricles was not increased." In the second experiment, a solution of carbonate of ammonia was injected into the veins of animals. Convulsions often very violent in character *instantly* ensued, and stupor quickly supervened. The respiration was difficult, the expired breath was loaded with ammonia, and vomiting of bilious matter occurred. While the stupor lasted ammonia continued to be expired, but when this disappeared the animals recovered their senses. What is the exact cause of the inflammations of the serous membranes, which often prove fatal in renal degenerations, does not seem made out; it does not appear to be the presence of urea in the blood, as we have no evidence that this is capable of producing such effects. Frerichs thinks that the impoverished state of the blood is an adequate cause, but in this we can hardly agree. While speaking

of urea as a substance abnormally present in the blood, it must not be left unnoticed, that it is only its presence in anything like considerable quantity that is abnormal, since it has been clearly proved that a minute quantity exists in perfectly healthy blood of men and animals. The same is the case with another constituent of the urine, viz: uric acid, which exists naturally in small proportion in healthy blood, but accumulates therein from defect in the excreting functions of the kidney just before an attack of acute gout, and also in chronic. As it is deficient in the urine, Dr. Garrod's conclusion seems just, that the chalk-like deposits appear to depend on an action in and round the joints vicarious of the uric acid excreting function of the kidneys. The well-known effects of the presence of this gouty matter (uric acid) in the blood, as the *malaise* and ill health which precede the attack, the inflammation produced by its localization, and the occasional serious result of its transfer to more vital parts, illustrate exceedingly well the disturbing action of an excretory substance retained in the blood.

Lactic acid was believed by Dr. Prout to be the *materies morbi* in rheumatism; he states that it is thrown off in immense quantities from the skin during the perspiration. Absolute proof of this, perhaps, is wanting, but as we know that this acid can be obtained from muscular fibre, and exists in the gastric, cutaneous, and urinary secretions, it is very probable that this is the case. In health, lactic acid is, most probably, rapidly disintegrated in the blood by oxidation, being converted into carbonic acid and water; in rheumatic and other diseases we may suppose this process to be interrupted, and that the acid, therefore, or its combinations, accumulates in the blood, and is thrown out by an excessive action of the perspiratory glands. This view is confirmed by the obstinate nature of many cases of rheumatism, and their great tendency to recur, indicating a deep-seated defect in some of the processes of organic life. It is also confirmed by the good effects of eliminative treatment succeeded by tonics, the object of which is to carry off the morbid matter that vexes the system, and afterwards to invigorate the general powers, so that the organic functions may be more properly performed.[1] Rheumatism is so manifestly akin to gout, that this conviction is a further argument for believing that the former depends, like the latter, on a *materies morbi*.

Whether *Bile*, when present in the blood, is the cause of disorders, we do not certainly know. Its gradual disappearance as it passes down the intestinal canal, is considered by Liebig as a proof that it is absorbed, but Lehmann is unable to find any trace of it in the blood of the portal vein. It may, therefore, be decomposed and not absorbed. Biliary pigment is often present in the blood in considerable quantity without occasioning much disturbance, but we cannot speak so positively with regard to the biliary acids. In that terrible affection of the liver called by Rokitansky acute yellow atrophy, in which the cells of the organ are completely destroyed, and the whole tissue deluged with yellow pigment, we are quite ignorant of the exciting cause of the convulsions and coma

[1] This view is the same as that most ably maintained by Dr. Fuller, in his truly valuable work on Rheumatism.

by which the disease commonly proves fatal. No chemical examination that we know of has yet been made of the blood in this disease, and all that can be said is, that it seems most probable that the poisonous matter which produces the cerebral symptoms is none of the constituents of the bile, since both of them, the pigment and the cholic acid, have been found in the blood when none of the symptoms of cerebral disturbance were present. It may be, perhaps, a decomposition-product of the organic biliary acids. However, though we cannot point out what the *materies morbi* in this case is, there is no doubt that the phenomena in this affection are owing to the presence of *some* abnormal matter circulating with the blood.

In that state of system which Dr. Prout has distinguished by the name of the *oxalic acid diathesis*, there seems good reason to believe that oxalic acid, or some of its salts, must be present in the blood, and be the exciting cause of the various symptoms. It is very probable that imperfect digestion often gives rise to the formation of this acid, but in other instances we are inclined to believe that its origin lies deeper, in a malperformance of some of the secondary assimilating processes. In not a few cases, the presence of this abnormal matter in the blood is betrayed by scarce anything else than the existence of characteristic octohedral crystals in the urine; but in others, and, perhaps, the majority, it seems impossible not to recognize a connection between the state of the blood evidenced by the urinary deposit and the peculiar nervous erethism and sensibility which exist. The circumstance mentioned by Dr. Prout, that those who have this diathesis are very liable to skin diseases, and affections of the nature of boils and carbuncles, also points to the presence of a *materies morbi* circulating in the blood. It is very conceivable, that a small quantity of this acid formed in, or introduced into, the blood, and constantly drained off by the urine, may give rise to no symptoms, but, that the presence of a larger quantity, and more especially its non-excretion by the kidneys, may cause great disturbance. This is in accordance with Dr. Walshe's experience, who remarks: "Observation continues to exhibit to us the frequency of a deposit of oxalate of lime crystals, at the period of convalescence of acute diseases; so much so that we regard their sudden appearance in an acute disease as a sign of that fortunate change. This deposit is of temporary (say a few days') duration, and not to be confounded with the more or less permanent condition appertaining to a peculiar diathesis."[1]

The above-mentioned substances, abnormally present in the blood, and producing disease, are tolerably well defined, but there are a multitude of others of whose nature we are totally ignorant, and which quite escape our means of observation. The principal of them are the infectious principles of the so-called Exanthematous diseases, including continued fever; syphilis belongs to the same category, and various cutaneous disorders, especially the squamous and vesicular. Variola, and its modification vaccinia, are the only instances in which we can at all pretend actually

[1] We have observed in a specimen of blood drawn from a man suffering under an attack of hemiplegia, a number of large octohedral crystals exactly similar to those of the urinary deposits.

to exhibit the *materies morbi*, and to transfer it from one system to another; even in these cases, the visible fluid is but the vehicle of the poison, for that is aeriform, and capable of being received through the channel of the lungs. The venom of deadly snakes, perhaps, may be an instance in which the matter inducing the morbid alterations in the blood of the bitten person is manifest and palpable, but even here we have no knowledge what the substance is which produces the septic effects. In the case, however, of deleterious gases, and of most poisons, the toxic agent is clearly known, and we can form some idea of its mode of operation. It would be quite beyond our province to attempt any detail of the various poisons and the effects they produce; we can only observe that they are all referable with tolerable accuracy to three heads, or to two of these combined, viz: (1) poisons which act as irritants, producing more or less irritation and inflammation of various organs; (2) poisons which act as sedatives, causing paralysis, more or less immediate and complete, of the nervous system; (3) septic poisons, which seem to annihilate the vital power, and induce rapid putrefaction of all the organic fluids and solids.

With regard to the action of poisons there are two fundamental ideas which it seems desirable briefly to refer to. One is, that when a minute portion of virus is introduced into the system, it appears to multiply itself immensely, as if it possessed the power of transforming healthy matter into its own noxious nature. Such a multiplication must take place when an unprotected person is inoculated with the matter of variola, the minute quantity of virus introduced reproduces similar properties in the contents of the numberless pustules which are formed all over the surface. The same is, doubtless, the case with all infectious diseases, and with syphilis. In the latter instance, it seems worthy of consideration whether the great difficulty of eradicating the taint from the system may not depend on the less degree of constitutional disturbance which the virus occasions, and its inferior tendency to eliminate itself by undergoing certain transformative changes. May not the action of mercury be chiefly to promote these changes, and so render the *materies morbi* more ready to be eliminated; this seems both consonant with what we know of its action, and supported by its superiority over other drugs which are capable of producing much more powerful excretory action. Were the action of mercury merely that of increasing the action of the several emunctories, syphilis ought to be curable as well by sweating, purging, and diuresis, which is not the case. On this view also we perceive the reasonableness of not salivating a patient profusely, but maintaining for a good while a mild but efficient alterative action. The conception now mentioned applies more particularly to certain irritant poisons, the second to those that are termed septic. When spongy platinum is placed in a mixture of oxygen and hydrogen gases, they quickly unite together and form water, the platinum itself undergoing no change. This is an example of what Berzelius named "catalytic action;" there are many similar known instances, and it is very probable that actions of this kind are by no means infrequent in the animal system. The solution of the food in stomach digestion is, probably, in part, dependent on a catalytic action, or one of a somewhat similar kind, in which the

peculiar organic matter called *pepsin* disposes the alimentary ingesta to undergo solution in the gastric acids. A minute quantity of the change-inducing substance is sufficient to cause the action to commence, and so it appears a minute quantity of virus is sufficient to induce septic changes in the blood with which it is mingled. The history of cases of death from the bite of venomous reptiles, of the most malignant fevers, especially scarlatina, and of the effects of the matter of glanders, shows that the essential and primary action of these poisons is to lower extremely the vital powers, and induce putrefactive changes in the organic fluids. If this action be not utterly overwhelming, the system takes alarm, and manifests resistance and reaction by setting up the inflammatory process; but this, it is quite clear, is only the secondary result of the poison, and not essential. In some constitutions the vital power is weak, and is seriously affected by comparatively slight agencies; thus, it is recorded that very alarming symptoms have been produced by the sting of a bee and of a wasp. The state of the blood when affected by septic poisons will be hereafter described under the head of Necræmia, an appropriate name which has been assigned to this state by Dr. Williams. Pyæmia is the name given to a certain state of the blood somewhat akin to the foregoing, in which an unnatural matter, that of pus, is present and circulates with it. The matter itself will be described hereafter when we speak of the products of inflammation, and the condition will be considered as a general disease of the blood.

ANÆMIA, SPANÆMIA.

There can be little doubt that by excessive hemorrhage, or exhausting discharges, the whole mass of circulating fluid in the vascular system can be considerably reduced; that is to say, the result of such losses is not only to impoverish the quality (as we know it does), but to diminish the quantity of the whole mass of blood. The term *anæmia*, signifying absence or deficiency of blood, is therefore correct, though if it were not so commonly received and employed, one might wish to substitute the term *oligæmia*. *Spanæmia* is the name proposed by Dr. Franz Simon to express a deteriorated quality of the blood (σπανος, poor); it almost always accompanies the state of oligæmia, or anæmia; both may, we are inclined to think, exist not unfrequently as the sole condition itself, the blood being of normal quantity, but impaired quality. We will here recapitulate shortly the changes which have been before detailed in the several constituents of anæmic and spanæmic blood. (1.) The red corpuscles are remarkably diminished, 127 being the average per 1000; they have been known to sink as low as 27; they also appear to contain less hæmatine, being somewhat paler than those of healthy blood. (2.) The amount of white corpuscles does not appear to be altered; in some of our examinations they have been found as numerous as in healthy blood. (3.) The fibrin is quite unaffected; it was never found below the normal mean, and in cases where inflammation of some organ was present, its quantity was notably increased. (4.) The solids of the serum have not been found specially altered.

(5.) The quantity of water is more or less increased in proportion to the diminution of the globules; in the case above mentioned, where the globules were only 27 per 1000, the water was 886.

The *causes* which produce anæmia and spanæmia are: (1.) Losses of blood, whether natural or artificial, the red globules being thus diminished, their place is supplied in great degree only by the absorption of water. (2.) Profuse discharges of watery, mucous, or albuminous fluids, such as occur in aggravated leucorrhœa, diarrhœa, or in cases of cauliflower excrescence. In these, it seems as if the blood-globules were melted down to supply the profuse drain; probably they perish, or are not reproduced from want of a proper nutrient fluid. (3.) Insufficient food; the effect of a greatly improved diet in increasing the amount of red corpuscles was very apparent in a case under our observation, in which iron had been previously administered, with some, but not marked benefit; while on the improvement of the diet the amelioration was rapid. Too often, no doubt, this cause operates powerfully in inducing the anæmia so common among young females of the lower classes. (4.) Deprivation of fresh air and light; the effect of this can scarcely be overestimated. Even the best food will not be converted into healthy blood if light and air are withheld; while a coarse and insufficient nutriment will not prevent a person from having a ruddy color, if he be much in the open air. Of this we have frequent instances among our laboring population. (5.) An unhealthy crasis of the blood, in consequence of which the existing blood-globules are imperfectly nourished, and the development of new ones is hindered. Such is the cause of the anæmia in persons suffering from degeneration of the kidneys, from lead cachexia, the cancerous diathesis, perhaps the tubercular, and in some chlorotic cases. There can be no question that in many cases, as is well described by Dr. Williams, the anæmia is not the cause but the result of the amenorrhœa. The suppression of the natural evacuation leaves the blood in an unpurified state, which is unfavorable to the development of healthy hæmatine. In the same way rheumatism may prove a cause of anæmia, and in several of Andral's cases, the globules at the first bleeding were found not to be below the ordinary average. Mental anxiety may probably also be considered as a cause of this kind.

The symptoms of anæmia and spanæmia depend immediately on the impoverished condition of the blood. The face is pale or sallow, according to the natural tint of the skin; the prolabia are blanched from their cherry red; even the tongue presents unnatural pallor. The conjunctivæ, it may be remarked, are clear, which should always be observed, and may prevent the error that has been sometimes committed of mistaking the sallow tinge of the complexion for a bilious, and directing the treatment accordingly. In some cases there is a show of patchy redness on the cheeks, but this is very different from the natural diffused redness which is seated in the capillary plexus, and rather seems to depend on the congestion of some superficial thin-coated veins, which naturally would not be seen.

To the ear the impoverished condition of blood announces itself by certain abnormal noises in different parts of the vascular system. These, the so-called inorganic murmurs, as distinguished from those

which depend on structural alterations, are commonly heard over the base of the heart, and at the root of the neck on either side. The first are produced at the origin of the aorta, or of the pulmonary artery, and are probably, as Dr. Williams believes, of the nature of ripples, the natural inequalities of the surface over which the current passes being sufficient to occasion in its dilute and diminished condition "vibrations and sonorous gushes," which would not occur in a fluid of greater density. The latter are generally believed, and no doubt correctly, to be seated in the larger veins; they probably depend, partly on the vibration of the valves, partly on local compression, which causes a sonorous gush, where the fluid passes from the narrowed channel into the wider.[1] It must not, however, be omitted, that these murmurs are not absolutely a sign of anæmia; they are also audible, though with less intensity, in many young persons, and in some aged, who present no trace of this condition. As the impoverished blood runs the round of the circulation, all the parts that are dependent upon it for the maintenance of their several powers become more or less injuriously affected. Those in which the process of decay and repair is most active, of course will suffer earliest and most. Accordingly, we find the muscular and nervous system the seat of most marked disorder.

The heart, illustrating the converse of the aphorism, that "repose is the revelation of power," betrays its feebleness by the weak and thready state of the radial pulse, by the coldness of the surface and extremities, and by the sudden, brief, often palpitating or irregular character of its contractions. The least bodily or mental excitement is sufficient to cause violent palpitation, as if the organ were conscious of its weakness, and strove by the frequency of its action to compensate for the imperfection of it. Sometimes, even without any excitement, the heart beats very forcibly, so much so, that an inexperienced observer might easily be led to suppose the organ hypertrophied; but the sharp knocking character of the impulse is extremely distinct from the steady, strong, heaving swell of the real hypertrophy. Such continued increased action of the heart is as much due to an abnormal condition of the nervous as of the muscular system. The contractile power of the muscles generally is impaired, and a slight effort induces fatigue, or even faintness; there is no capacity for any sustained exertion; the bowels are often costive, apparently from want of tone in the muscular coat, which should propel the contents onward. The disorder of the nervous system is especially manifested by the increase or perversion of the natural sensibility. All causes of pain or uneasiness produce more than their usual effects; a variety of anomalous distressing sensations are complained of, some of which are fugitive, or affect one part after another, but one at least locates itself with remarkable constancy in the left side or hypochondrium. It is difficult to say what is the real state of the morbid action that occasions these pains, whether it be in the central organs, the pain, according to a well-known law, being referred to the peripheral termination of the fibre, or as we are rather inclined to think, located in the nerves themselves, their delicate substance being

[1] Kivisch, however, earnestly contends for the arterial seat of spanæmic murmurs.

in some degree disordered by the imperfect nutrition afforded by the impoverished blood. The sympathetic nervous system is also affected; the appetite is lost or sometimes strangely perverted, so that the patient will eat chalk, cinders, sealing-wax, &c.; the stomach becomes irritable, and often cannot tolerate substantial food or tonic remedies, or, together with the intestines, secretes enormous quantities of gas. The nerves of the kidneys are often so affected, that the secretion of the organs is materially modified for a time, and a copious flow of almost aqueous urine takes place. Or, those of the bladder may be affected, and there may be either inability (supposed) to void the urine, or to retain it. The nerves of special sense may be affected, and intolerance of light and sound, flashes before the eyes, and noises in the ears, may be present. The intensified action of the heart has been already referred to. All these symptoms indicate that condition already described, in which the sensibility of the incident nerves themselves appears increased, and the receptive and reactive power of the nervous centres also. The nervous system in the anemic condition may be likened to a spring, which originally was of a certain strength, requiring a certain impressing, and reacting with a corresponding force, but having become much weakened, is bent by a much less force, and reacts also with much less. Mobility and debility may be said briefly to be the chief characteristics of the nervous actions in the anæmiated. Dr. Williams, remarking on the nervous excitement of anæmia, and contrasting the (apparent) increase of this function with the failure of others, is inclined to account for it by the circumstance, that the encephalic bloodvessels, being less exposed to atmospheric pressure than the vessels of other parts, are apt to contain relatively more blood then, under the circumstances, than they should. This undue supply of blood, if the heart's action be hurried, or excitement be otherwise induced, may produce an erethism of the nervous centres, with the symptoms above mentioned; or if the heart's action be languid, it will stagnate, and occasion headache, relieved by the recumbent posture, drowsiness, impaired mental faculties, or even, in extreme cases, coma. The stagnation probably takes place chiefly in the large veins and sinuses. We confess that we doubt the correctness of Dr. Williams's fundamental assumption, that the encephalic bloodvessels, in the anemic condition, contain more than their due share of blood. We have seen so often in autopsies the most marked pallor of the membranes, and emptiness of the bloodvessels, except the large venous trunks, that we cannot think the peculiar position of the vessels exempts them at all from being in a like condition, as to fulness or emptiness, with those in other parts of the body. In fact, the sub-arachnoid fluid is to them what the atmospheric pressure is to others; and hence an anemic brain is commonly a "wet" one—*i. e.* the sub-arachnoid fluid is increased. The real cause, we believe, of the nervous excitement which is apt to occur, is the altered condition of the nervous matter, both white and gray, in consequence of its defective nutrition. Hence (like the weakened spring) it becomes so susceptible, that it is injuriously affected by even natural and healthy excitants. Perhaps, also, deficient tonicity of the vessels may contribute, in part, to increase the cerebral excitement. This would allow of an increased

flow of blood to the brain, and also make it be attended with throbbing, on account of the flaccid state of the conducting pipes.

Weak digestion, or apepsia, is a very frequent accompaniment of anæmia, and is doubtless occasioned by the debilitated state of the muscular coat of the stomach, as well as by the deficiency of gastric juice, which the follicles are unable to furnish, in consequence of their own nutrient supply being defective. Hence arises a further cause, which continues and aggravates the anæmia. Healthy chyle cannot, of course, be formed to renovate the blood, if the digestive function is seriously impaired.

Some of the results of anæmia may next be noticed. Several instances have occurred in which the nutrition of the heart had suffered so much, and the organ become so debilitated, that sudden and fatal syncope was the result. The possibility of this should always be borne in mind in treating a case of severe anæmia, and the patient should be enjoined to avoid sudden efforts, and to remain as quiet as possible until some degree of strength and tone is restored. It is a question of much interest, but as yet we believe not determined, whether the increased action of the heart above described ever produces structural change. It would be thought likely that a weakened hollow organ, contracting repeatedly for a long time on a mass of blood poured into it, would be apt to yield somewhat to the outward pressure or resistance of the fluid, and thus become dilated; but it does not seem to have been shown that this actually takes place. Some degree of anasarcous swelling of the feet and ankles is not uncommon. When this occurs, we may take it as a sign, on Andral's authority, that the albumen of the serum is diminished. Asthenia may advance to such a degree, that it proves fatal by a gradual failure of the vital powers, like the sinking at the close of diseases of exhaustion. The impoverishment of the blood may probably be the determining cause of the appearance of tuberculous or other cachetic diseases. Dr. Williams describes a fatal termination to anæmia, which we have not actually witnessed, although we have seen more than one occurrence so closely similar, that we are convinced of the correctness of his account. The importance of the subject makes us unwilling to abridge Dr. Williams's description: "A young female becomes anæmic; and after exhibiting various symptoms of feeble general circulation, with headache, drowsiness, and impaired sensorial functions, suddenly becomes worse; passes into a state of stupor, with dilated pupils, sometimes varied by slight manifestations of delirium, throbbing of the carotids, and partial heat of the head, and dies comatose. On opening the head, a small quantity of serum is found under the arachnoid, and in the ventricles, sometimes with a little lymph (in one case there was none). The vascularity of the membranes is remarkable, but the vessels most distended are the veins, and in the larger of these, and in the longitudinal sinus, there is a firm coagulum. In parts, especially at the torcular Herophili, this coagulum blocks the whole sinus, and exhibits a separation of fibrin, portions of which are softened down into that opaque purilaginous matter, which was long mistaken for pus, but which Mr. Gulliver has shown to be a mere disintegration of the fibrin, which mere stagnation in a warm temperature may effect. These have been taken for cases of meningitis. No

doubt inflammation may supervene in them occasionally; but in two cases that have fallen under my notice, there was no adhesion of the arachnoid, nor deposit upon it, nor any other unequivocal mark of inflammatory action; yet the fibrinous and bloody concretions in the veins and sinuses were most remarkable for their size and firmness."

In the cases of similar nature which have fallen under our own observation, the veins of the lower extremities, one or both, have been affected. Owing to the kindness of Dr. Cursham, we had lately an opportunity of examining a very marked case of this kind. It occurred in a youth who died in the Brompton Hospital with empyema of the left side, and tubercular disease of both lungs. The body was emaciated, exceedingly anæmic, both lower limbs highly anasarcous, and traversed here and there by superficial veins, distended with dark blood, and feeling hard and cordy. One limb had first become anasarcous, and afterwards the other; no symptoms of phlebitis appeared to have existed. The lower part of the vena cava inferior, and all the veins below, as far as they were traced (to below the knee), were blocked up by coagula, of more or less decolorized fibrin. In many places, the exterior layer of the coagulum had almost assumed a membranous appearance, and was very closely adherent to the wall of the vessel. It could, however, be completely detached by a little care, and then the coats of the vein appeared perfectly natural. They had their normal elasticity and firmness, and were not even for the most part stained with blood. It was evident that their tissue had not been inflamed. The fibrinous coagula in several places were somewhat softened, and rendered slightly spongy or reticular in their interior. This was due to a spontaneous transformation taking place in the mass. Instead of presenting a dense network of fibrils, set in an hyaline substance, interspersed with a little granular matter, and with a few corpuscles, which was the case with the outer layer of a coagulum found in the right ventricle, the softening fibrin from the interior of the coagula in the large veins consisted of an immense number of various-sized corpuscles, some quite similar to glomeruli, imbedded in a mass of granular and oily matter, with scarce any trace of the fibrillar network. This change in the fibrin was evidently not such a mere disintegration as a warm temperature might occasion; had it been complete and general it would have led to the breaking up of the coagula, and the restoration of the circulation. In other instances that we have seen, under the influence of appropriate treatment, this actually took place, and all obstruction disappeared. It is very conceivable how anæmic blood, with its vitality generally lowered, and especially deficient in the organized living cells, shall tend, particularly when it is propelled in a sluggish current by a languid heart, to pass into that condition which it spontaneously assumes when withdrawn from the influence of the living tissues: and it is very manifest how important it is to be aware of the tendency which exists to such an event, and, if it occurs, to appreciate its real nature, and not to regard it, as might easily be done, as the result of inflammation. The nutrition of some parts in the anæmic may be impaired to such an extent that ulcerations form spontaneously. The cornea would appear especially likely to suffer in this way from not being permeated with vessels. Such instances of morbid action are very important to notice, as being free

from complication, and exhibiting, therefore, more clearly the essential nature of a process. Ulceration in this case is clearly not produced by inflammation. It is rather interesting to remark, that the most lowly organized and the least essential of all the tissues, viz: the adipose, suffers less from impaired nutrition in the anæmic state than any other. It is by no means uncommon to see persons, especially females, presenting a considerable amount of *embonpoint*, who are manifestly very deficient in healthy blood. This is the more easily comprehensible, as the fat vesicles really seem to be scarce more than so many minute drops of exuded oil, included in homogeneous films of protein material.

The foregoing history of anæmia and spanæmia manifestly relates to it as a *general* condition. It seems very doubtful how far there can exist such a condition as partial anæmia, if we recognize a deficiency of red corpuscles as an essential feature of this state. Of course, the supply of blood to a part may be defective in consequence of various causes, but this does not involve any alteration in the quality of the fluid transmitted to the part. However, using the term in the sense of merely deficient supply of blood, the consequences of such a state will be generally those of diminished nutrition, or, more properly, atrophy of the part, with more or less considerable impairment of its function. If the deprivation of blood be very great, mortification may be the result; this has occurred in some cases in which the main artery of a limb had been tied on account of aneurism, and the collateral circulation did not establish itself soon enough. Even in cases which have a more favorable issue, the immediate effect of cutting off the supply of blood is to occasion weakness, numbness, and reduction of the temperature; the muscles and nerves are, in a great measure, paralyzed, and the heat-producing process fails with that of nutrition. When the aorta of an animal is tied, its lower extremities, after a time, become as paralyzed as if its spinal cord had been divided. The causes of local or partial anæmia may be, (1) tumors of various kinds situated so as to press upon and obstruct the main artery supplying the part; (2) disease, often atheromatous, of the coats of the vessel itself, leading to deposition of fibrinous coagula, and consequent obstruction of the channel; (3) spontaneous coagulation of the blood in an artery; (4) blocking up of a vessel by fibrinous flakes transported from a distance, perhaps from the valves of the heart; (5) withdrawal of the nervous influence from a part, in consequence of which its nutrition fails.

It may be observed, that it is difficult in many cases to say positively whether anæmia of an internal organ exists; we cannot observe during healthy life what amount of blood as indicated by its color it contains, and the changes in the distribution of blood which may ensue during the last hours of life, and after death, will greatly alter the natural appearance.

HYPERÆMIA.

Hyperæmia, the opposite condition to anæmia, implies, of course, an excessive quantity of blood. The term is commonly applied to accumu-

lation of blood in a part, *i. e.* to local or partial excess, while plethora (πλῆθος, a multitude) is that which is used to signify increase of the general mass. We will first consider plethora, or general hyperæmia, and afterwards partial.

The characters of marked plethora are strongly expressed, and easily discernible. The face is rather full and turgid, and presents a diffused redness, often of a slightly purplish tint; this is especially observable in the lips. The conjunctivæ are redder than natural, the expression of the eye sharp and ferrety. The pulse is full, and more or less strong. The temperature of the skin is inclined to be hot, and even in the most remote parts it is fully maintained. There is a tendency to headache, and not unfrequently there is some degree of drowsiness and disinclination to exertion. Persons in this state have good appetites, and digest their food well, the secretions all seem to go on naturally, and organic or vegetative life is in full vigor. They often lead sedentary lives, in consequence of which the waste of the tissues is diminished, and the plethoric state augmented, but this is not constant. In many, a considerable amount of adipose tissue is formed, which, as Dr. Watson remarks, may serve as a kind of safety-valve for the diversion of the superfluous blood; no doubt this is true; and we are inclined to think that the peculiar symptoms of plethora are most marked when there is no remarkable accumulation of fat. On account of the increase in the quantity of blood it is manifest that complete oxygenation of it must be more difficult; hence, on any exertion the breath is apt to be short, and the action of the heart laboring; hence, also, as Rokitansky observes, the blood always presents a certain degree of venosity, as if never thoroughly arterialized. A very just distinction is made between two principal varieties of plethora, the sthenic and the asthenic. Dr. Williams (whose description of them is most excellent) considers that the difference between them depends chiefly on different proportions of contractility and tonicity; that is, on the different degrees of the vital endowments of the heart and vessels. Doubtless these are increased in sthenic, and diminished in asthenic plethora, but we cannot but believe that there are other differences also. The quality of the blood in all probability is different, and the vital properties of the other organs are also different. Generally, it may be said, that in the one the organic life and tone of all parts is exalted, in the other proportionately depressed, while in both the mass of blood is in excess. Heat of skin, frequency (not, however, great) of pulse, with fulness and hardness, keen sensibility, mental and bodily activity and energy, a tendency to gout, bilious attacks, and disease of sthenic type, characterize the first form of plethora. It is observed in the "young, the active, and those of sanguine temperament." "Its tendency," according to Dr. Williams, "is to cause general febrile excitement, active hemorrhages, fluxes, and inflammations." Dr. Watson, on the other hand, remarks that the subjects of plethora are not, as they might naturally be supposed to be, and as many writers state them to be, peculiarly prone to suffer inflammatory complaint. "There is general fulness of the vascular system, but no irregularity, nor any necessary tendency to irregularity, in the distribution of the blood." We are inclined to think Dr. Watson's opinion the more correct of the

two. In asthenic plethora the skin is cool, the extremities apt to become cold; the pulse is large, but without resistance; it is often slow, sometimes irregular. The venosity of the blood is marked, the lips are often of a livid tint. The contractility and tone of the muscles is deficient, the spirits depressed, the mental and bodily activity diminished. It is most often seen in the aged, in those who are exhausted by excesses or previous disease, "or in whom the excreting organs act imperfectly." This latter condition, involving the imperfect depuration of the blood, is not a cause of the plethora, at least not to any great degree, but rather of the asthenia modifying the plethora. The tendency of asthenic plethora, Dr. Williams says, is to produce congestions and passive hemorrhages, fluxes and dropsies, and if continued, structural changes in some organs, as dilatation of the heart, enlarged liver, varicose veins, &c. Most of these effects, in our opinion, imply a further alteration of the crasis of the blood than belongs to uncomplicated plethora. What has been termed "excrementitious plethora," seems to be nearly the same as asthenic plethora, with impaired action of the excreting glands; this, Dr. Williams thinks, may arise from mere stagnation, or imperfect motion of the blood, in consequence of which "it becomes loaded with urea, lithic and lactic acid, and other effete materials, which unfit it for its proper uses, and irritate and disorder the organ through which it passes." A more likely cause of such a state of blood we believe to be the existence of unobserved organic disease of the kidneys or other glands. The characters of the blood in sthenic plethora are: (1.) The increase and amplification of the entire mass. Of this we have no direct measure, but we may form a tolerable idea of the extent to which it takes place, by observing the effect of bloodletting. As much as forty or fifty ounces may be drawn at once in some cases without fainting being produced, and even this quantity has sometimes been exceeded. Dr. Watson mentions a case in which seventy-two ounces were withdrawn before the patient became faint. Not only does the system *tolerate* these large losses of blood, but judiciously employed they are highly beneficial; the patients are relieved and refreshed by taking off a part of the mass which loaded the vascular system. So conscious are they of this, that those who "make blood fast," as the popular phrase is, will come and request to be bled, often at the spring of the year, when the blood-making process seems to go on more actively. (2.) The red globules in plethoric blood are remarkably increased, while the fibrin rather inclines to be somewhat diminished, and the albumen of the serum undergoes little variation. The quantity of water being diminished in proportion to the increase of the red corpuscles, it follows that the coagulum formed after bleeding will be large, and will be surrounded by but little serum. The mass of corpuscles in proportion to the fibrin is so great, that the latter cannot contract to the degree it ordinarily does, and hence a larger amount of serum is retained within the clot. In sthenic plethora, the coagulum is firm as well as large, in asthenic, its cohesion is diminished. (3.) A tendency to deficient arterialization may also be mentioned as a character of plethoric blood; perhaps we may connect this with a deficient production of fibrin, which, as we before stated, may with much probability be regarded as an oxidation product. Should this be the

case, there would appear some ground for accepting the following view, which is only offered as a suggestion.

The perfectly homogeneous aspect of some of the casts of the renal tubules which are found in the urine when fibrinous fluid has been draining off from the congested bloodvessels, suggest the possibility that fibrin may be particularly applied to the formation and renewal of the various homogeneous membranes, such as the limitary membrane of gland tubes, the sarcolemma of muscular fibres, the wall of capillary vessels. If then in some cases of plethora (the extreme ones) the fibrin is much diminished, it is very conceivable that the walls of the capillaries are less perfectly formed, and consequently less able to resist the interior pressure of the amplified mass of blood. This would, of course, favor the occurrence of hemorrhages, especially such as capillary apoplexy in the brain.

Among the *causes* of plethora, the first place, perhaps, is to be assigned to a special tendency innate in the system to form an undue quantity of blood, or, speaking more exactly, to a too rapid growth and multiplication of the red corpuscles. This, as it requires, so it may produce an increased quantity of Liquor Sanguinis, according to the principle that the demand induces a supply. When the tendency to form blood is considerable, it will manifest itself even in spite of circumstances that oppose it; but a similar tendency, in much less degree, will produce a most highly plethoric state, if favored by an ample supply of rich food and a sedentary life. Indeed, these may have the same effects, even supposing no predisposition to plethora at all to exist. It is worth remarking, however, that they will not produce this result in all cases. In many it would be rather dyspepsia, or some cutaneous disorder, or a bilious attack. Most of the circumstances that promote a robust state of health, with the exception of exercise, are favorable to plethora, and on the other hand, such as depress the general vigor, or induce diseases of debility, prevent its development.[1] Asthenic plethora is probably in most cases dependent upon an unhealthy state of the Liquor Sanguinis, occasioned by impaired action of some of the excretory glands, which itself may depend on some latent organic diseases of the same.

The *consequences* of plethora have already been in part alluded to. They are generally such as result from over-distension of the vascular system. On account of its proximity to the heart, its delicate structure, and the large supply of blood it receives, it is not surprising that the brain should suffer from this cause more than most other organs. Rupture of some of its thin-walled vessels may take place, or the blood be poured out from numerous capillaries; and this, of course, will be still more likely to occur, if another consequence of plethora be present, viz: cardiac hypertrophy. It is easily conceivable how the increase of

[1] There is no doubt that the cessation of habitual discharges, or their arrest by art, especially when suddenly effected, and without any corresponding modification of the system, induce a dangerous plethora. This should never be forgotten, not only in treating persons who are manifestly of plethoric habit, but even those who seem and are really in a different state. It seems that the vascular system, after having been long insufficiently filled with blood, cannot bear the amount of distension immediately which in the state of health would only be natural to it.

the mass of blood shall require and gradually induce an augmentation in the power and capacity of the organ that keeps it in movement. In sthenic plethora the hypertrophy will be more pure and simple, in asthenic it will be associated, in a greater or less degree, with dilatation. The great capacity of the vascular system of the liver will cause it to be enlarged by the increased distending force of the blood mass, more, in proportion, than many other organs. This will be especially the case if dilated hypertrophy of the right chambers exist, and the blood is thrown back on the venous side of the circulation. Again, in consequence of hepatic congestion, the tributaries of the portal vein will also be congested; and this seems more especially to affect the hemorrhoidal plexus of veins, which become distended into the little tumors, well known as "piles," and often give rise to a salutary hemorrhage. Another hemorrhage, not unfrequent in plethoric persons, especially the young, is from the veins of the nose. This seems especially to give relief to cerebral congestion. Menorrhagia may also be dependent on, or at least greatly increased, by a plethoric state. The natural determination of blood at the catamenial periods will of course be often attended with a greater discharge, on account of the increased tension of the vascular system. Though we consider it at least doubtful whether the plethoric are more prone to inflammation than others, there is no doubt that when inflammation is set up in them it is more violent, and requires more active treatment. Dr. Copland states also that the severer forms of inflammatory fever in the West Indies affect young and plethoric strangers rather than older residents, the aged, and the weakly. Bilious, gouty, and renal disorders, especially such as belong to the lithic-acid diathesis, are often considered as proceeding from plethora; but it may, perhaps, be questioned whether they do not rather take their origin in the causes of the plethora itself, the high feeding, insufficient exercise, &c. It is not improbable that plethora may play some part in producing a varicose state of the veins, but it is very doubtful whether aneurismal disease is ever occasioned by it.

We must remark with regard to Local Hyperæmia, as we did with respect to local anæmia, that it differs from general hyperæmia, or plethora, not only in the less extent to which it exists, but in not involving any qualitative alteration of the blood. Local hyperæmia, in fact, is not exactly local plethora; it simply implies that too much blood is accumulated in the vessels of a part, without taking any count of the nature of this blood. Hyperæmia of a part is a phenomenon which naturally attracts attention, and has been considered and commented on from the earliest times. It exists in the most various conditions, from that of increased vital power and functional activity of an organ, to that of cessation of all action in it, its death and decomposition. One of the best examples of a physiological and natural hyperæmia is afforded by the female breast during the period of lactation; the vessels proceeding to it enlarge considerably, and it manifestly receives much more blood than at other times. So great is the flow of blood to the part, that it not unfrequently happens, owing to a deficiency of secretory power, that the healthy hyperæmia becomes excessive, and a cause of

inflammatory disease. The limits of physiological hyperæmia are pretty wide. It is often striking to observe how much more blood is contained in the vessels of a part that is actively employed, than would be present there under ordinary circumstances. Indeed, there is much to lead one to the belief that it is not so much the amount of hyperæmia that determines the transition from the healthy to the morbid state, as the alteration of the vital condition of the tissues of the part. In endeavoring to study the various conditions under which hyperæmia occurs, we cannot do better than adopt the arrangement proposed by Dr. Williams, and consider, *first*, hyperæmia with diminished motion of the blood in the part; *second*, hyperæmia with increased motion; *thirdly*, hyperæmia with motion partly increased, partly diminished. The first of these states may be designated *congestion;* the second, *determination of blood;* the third, is *inflammation*. This arrangement has the advantage of classing together several conditions, in which hyperæmia is a prominent phenomenon; but it is not certain, especially as respects determination of blood and inflammation, that it is correct; or rather, that it may not prove a source of error by not being founded on that which is the essential circumstance in these two conditions. This, at present, is not possible, from the imperfection of our knowledge; but it may be well to bear in mind the above caution.

Congestion—employing the term to signify excess of blood in a part with diminished motion—affects chiefly the small veins of the part, and the capillaries that communicate with them. Hence, its color is inclined to be of a dark venous tint, unlike the more vivid blush of inflammatory redness. The part often exhibits patchy, irregularly-distended vessels, which can be emptied by pressure, but gradually fill again. Its temperature is not much, if at all, increased; and the pain felt in it is rather aching or dull than acute. The degree of swelling varies according to the cause producing the congestion, and other circumstances; generally it is not very great. Congestion may exist alone, but often there are present also some exudations, the results either of it, or of the condition which gave rise to it.

The two principal causes which produce congestion are: (1.) Obstructions of various kinds to the return of blood through the veins. (2.) A relaxed and toneless state of the capillaries and small veins. Of the first cause we have a good example in tying up the arm for venesection; the current of blood setting towards the heart being obstructed, and the artery continuing still to pour in fresh quantities, the capillaries and all the veins up to the obstructed part become distended with blood. This is marked by the red or purple color of the part, and its swollen condition. The same effects will of course be produced in all instances where the veins of any organ are obstructed; obstruction of the jugular veins produces congestion of the brain, of the renal veins, congestion of the kidney, and so on. The modes in which the obstruction may be produced are very various; to take the brain as an instance, the veins returning the blood from it may be pressed on by an enlarged thyroid, or by a mass of indurated glands, or by an aneurismal tumor. Temporary cerebral congestion may also be caused by a prolonged expiration, or by holding the breath, especially when muscular exertion is made at the

same time. The arrest of the blood in the veins in these cases depends partly on the diminution of the capacity of the chest which takes place during expiration, partly on the suspension of the respiratory movements, which cause the blood as well as the air to rush into their respective cavities within the thorax during inspiration. Perhaps the very most marked instance of the effects of this arrest is manifested by severe cases of hooping-cough. The capacity of the chest is narrowed more and more by the repeated expirations, and the blood not being drawn onwards, but thrown back, accumulates visibly in the face, which becomes turgid; in the eyes, where ecchymosis sometimes takes place; and similarly, though we cannot see it, in the brain, where extravasation may also occur, or such congestion as produces an attack of convulsions. Obstructive valvular disease of the heart, throwing the blood back on the lungs, is the cause of the abiding dyspnœa which characterizes such complaints. The condition of the large hepatic veins, and of the inferior cava where it receives them, shows that the influence of inspiration must be felt as a powerful cause in promoting the circulation through the liver; when this, therefore, is impaired, as is the case in vesicular emphysema, or other diseases interfering with the respiratory movements, the liver will be congested, and the same will of course occur when in consequence of asphyxiating causes the blood does not pass freely through the lungs, but accumulates in the right side of the heart, and in the large veins. Now, in all such instances of congestion it may be remarked that there is no evidence of excitement of the part, if we except, at least, the occurrence of convulsions in attacks of paroxysmal cough; the temperature of the part is not raised, the functional activity is rather diminished than increased, and exudations from the bloodvessels, if they occur, show no tendency to organization. Everything indicates that the hyperæmia is merely the result of a mechanical cause, and that there is no primary and special alteration of the vital endowments of the part. The second cause of congestion, viz: atony of the vessels, may occur either primarily or secondarily. In adynamic fevers, in states of extreme debility, and perhaps in some persons whose tonicity is naturally defective, the vessels of a part become distended with blood, without any obstruction existing in the veins which convey their blood away, or without any previous inflammation or undue excitement having exhausted their natural contractility. The whole surface of the body, in some fevers of very low type, is covered with patches of congested vessels, and it is to be noticed that these are chiefly seated in the under parts, the blood gravitating downwards, and accumulating in this situation. Hence we derive a hint for a precaution well worth observing in continued fever, viz: to alter the position of the patient occasionally, and not to allow the blood to gravitate day after day to the posterior parts of the lungs, which in consequence are especially prone to engorgement and hepatization. Primary atony of the vessels of the choroid coat of the eye seems not unfrequently to occur, and to be the cause of the muscæ volitantes to which the dyspeptic and others are subject. The vessels of the uterus in passive menorrhagia, and those of the vagina in non-inflammatory leucorrhœa, are not unfrequently affected by primary atonic congestion. It is, however, much more common that atony of the vessels occurs

secondarily in consequence of some previous excitement or inflammation. This, in fact, is almost always the case in persons of feeble power when they are attacked by inflammation; when the disease is subdued there still remains behind this congested state of the overstrained vessels, which being naturally of weak tonicity, are unable to resume readily their proper caliber. In persons of more vigorous constitution, the original tonicity being greater, the vessels quickly recover their normal dimensions as soon as the strain arising from determination of blood is diminished; hence convalescence is speedy, and the restoration of the part complete. But when the system is naturally feeble, or when injudicious treatment has rendered a strong system so, then it may be a most difficult task to revive the contractility of the languid vascular coats, and remove the congestion which necessarily ensues. A healthy person may have an attack of acute bronchitis, or pneumonia, and if he is properly treated he will recover completely, and be scarce any more liable to the disease than if he had never suffered at all. But how different is it when chronic bronchitis is set up in the aged or debilitated; the disease commences generally without very severe symptoms, the inflammation does not run high, it may be soon in great measure subdued; but after that is accomplished, the capillaries of the bronchial mucous lining are unable to resume their tone, or they resume it partially, and easily lose it again when they are exposed to the slightest strain, and so the congestion occurs over and over again, until the vessels become mere flaccid channels gorged with slowly-moving blood, and pouring out exhausting exudations of muco-purulent fluid. It is very apparent that the longer the vessels remain congested, the more difficult it must be for them to recover their normal dimensions, and hence we derive the valuable hint to subdue active inflammation in every instance as quickly as possible, that the strain upon their walls may be lessened, and when this is accomplished, to turn as soon as is prudent to remedies of astringent and tonic character. This condition of atonic congestion is often seen in the conjunctiva after it has suffered an attack of acute inflammation, and we can scarce have a more valuable lesson than the observation of such cases, and of the *lædentia* and *juvantia* offered us. A case of this kind is related in Mr. Tyrrell's work; vol. i. p. 24, which from the first time we read it has never passed from our mind; and often has the valuable instruction it conveyed been the means of directing us to successful treatment. It shows how, besides the local signs of atonic congestion, the general condition of the system is also to be considered, and that, if this be found in an enfeebled state, no means will be nearly so efficacious in removing the congestion as those which impart tone and vigor to the tissues generally, and to the vessels in particular.

Besides actual inflammation, over-use of a part may occasion congestion of its vessels. Of this we have an instance in the congestion of the choroid, which is so common in those who exert their eyes very much upon minute objects. An organ which has been secreting with unusual activity is sometimes found congested with blood. This we have observed in the kidneys of diabetic persons; however, it is not constantly the case by any means. When it occurs, it is presumable that

the continued flux to the part had weakened, by the strain it caused, the contractility of the vessels. The influence of cold in producing congestions of internal organs cannot be doubted. To this we must ascribe, in part, the prevalence of chest affections during the colder part of the year, the blood being repelled inward from the surface by the constringing effect of the cold upon the vessels. The same cause also must be the chief agent in occasioning attacks of apoplexy, which have been observed to be greatly more frequent during a very cold season than during a mild one. The effect of the malarious poison in producing congestions of the internal organs is still more potent than that of cold. With every paroxysm the liver and spleen become greatly distended during the cold stage; and to such an extent may this take place, that the latter organ, extensible as it is, has been ruptured, and fatal hemorrhage ensued. Posture is a very efficient cause in producing congestion. It has already been alluded to, when instancing the pulmonary congestions that occur in fever; but the most marked examples of its effects are seen in the lower limbs. Here the returning venous current has to overcome the force of gravity, and though while the vessels maintain their tonicity, and the valves of the veins are efficient, this retarding force does not produce any effect, yet when the conditions are altered it becomes speedily manifest. Persons of feeble constitution, who are obliged to remain for the greater part of their time in an upright position, laboring hard and living poorly, are exceedingly liable to a congested, thickened, and indurated state of the integuments of the lower part of the legs and feet. The veins proceeding from these parts—and especially the large superficial veins of the limb—are seen tortuous, enlarged, distended, and varicose. They are evidently gorged with slowly-moving blood, the column of which, greatly enlarged in bulk, has much more difficulty in resisting the force of gravity than in the natural state. Such congestions proceed very soon to ulceration, which is apt to assume a sloughing form, and which can only be healed by means which bring about a more healthy circulation. These are, of course, directed to take off the force of gravity by the recumbent posture, to empty the distended vessels, and to supply, by external equable pressure, the defective tonicity of their walls. It may, perhaps, be questioned, whether the class of congestions from this cause should not have been included under those arising from venous obstruction, as the condition of the vein itself is such as to create an obstruction to its own current. Withdrawal of nervous influence from a part is sometimes the cause of congestion taking place in it. Mr. Simon records a case in which, after the ulnar nerve had been torn across at the inner condyle, the two inner fingers of the hand of the same side "had become swollen and livid with vascular injection." Disease of the trigeminal nerve, destroying its functional capacity, has often been observed to occasion inflammation and ulceration of the eye, and in some cases of the parts adjacent. This inflammation, it is most probable, was rather of the nature of atonic congestion, at least at its outset. It is not, however, to be concluded that the walls of the vessels lose their tonicity whenever the nerves of a part are unable to discharge their functions, or that this is actually dependent on nervous influence. No doubt it is the special

endowment of the vascular membranes, and only capable of being affected by the action of the nerves, as well as by direct stimulus. The effect of an atonic state of the walls of the vessels is well illustrated by an experiment performed by Dr. Williams: he adapted to a syringe a tube with two arms, one of which was connected with a metal tube, and the other with a portion of dog's intestine, of the same length as the metal tube, but, when distended, double its diameter. Water was now thrown in by the syringe, and the quantity discharged from the open ends of each of the two tubes estimated. The metal tube in the same time yielded three times more liquid than the intestine. Now a vessel, whose wall is possessed of a proper degree of contractility, may be compared to the metal tube. The force of the heart communicated by the fluid to its walls is not lost, but reacts again immediately upon the fluid and drives it onward. The vessel, with atonic flaccid walls, resembles the intestine, which yielded to the distending force of the column of fluid, and, from not reacting upon it, allowed a great part of the force to be lost as an impelling influence.

The effects of congestion have already been partially noticed. They have reference mainly to two circumstances: one, the impairment of the vital actions of the part, the other, the effusion from the overloaded vessels of watery, albuminous, or mucous fluids. When the arm is tied up for venesection, a sensation of numbness, weakness, and chilliness, is felt after a time, showing that the sensibility and contractility of the limb are impaired by the congestion which has taken place. In the hepatic and renal congestions, which often are produced by obstructive disease of the heart, the secretion of bile and of urine is commonly diminished, or morbidly affected. Cerebral congestion interferes materially with the free exercise of the functions of which the brain is the instrument. This impairment of vital (*i. e.* special) power depends partly on the more increased quantity of blood in the part, which, being greater than is proportionate to its functional activity, overloads and oppresses it. If it be true—as we shall see reason to think that it is—that the supply of blood to a part is, in a measure, dependent on the vigor and energy of life which that part possesses, it will not be difficult to conceive that an over-supply of blood will have an injurious and depressing effect on the same vital powers. But a still more powerful, and quite unquestionable, cause of vital depression in a congested part, is afforded by the altered condition of the blood itself, which, semi-stagnating in the capillaries and veins, becomes more venous than it should, and otherwise unfit for the healthy nutrition of the tissues. The sloughing ulcers which form in the congested and thickened integuments of the lower limbs, when the veins are enlarged and varicose, are a striking instance of the lowered condition of the vitality of those textures. Long-continued congestion of the liver, from disease of the heart, produces a very remarkable effect on the parenchymal cells. They become very greatly loaded with yellow matter (which does not appear to be true biliary, but rather bile pigment); in extreme cases, the majority atrophy, and are reduced to a mere granular detritus, while the capillary inter-cell-spaces become enlarged. These changes afford some explanation why the secretion of bile is interfered with, and also why jaundice

occurs. The effusions that take place from congested parts are certainly the most prominent phenomena of the condition. They will be most abundant, as a general rule, when the congestion depends on venous obstruction; so that, while fresh blood is being poured into the part, no exit can be found for it, except that which the exudation affords. They may also be extremely abundant in some cases, in which not only the tonicity of the vessels is entirely lost, but the texture of their walls is altered, so that they no longer oppose any obstacle to the escape of their contents, but allow them (the fluid part) to transude with great facility. Such is, no doubt, the case in instances of bronchorrhœa, chronic diarrhœa, and leucorrhœa, where large quantities of fluid are continually passing off from the toneless vessels, but where no venous obstruction exists, or none that is commensurate to account for the discharge. The effect of remedies in these cases shows that the discharge is dependent on the cause we have mentioned. Under the administration of turpentine or astringents the vessels regain their tone, and no longer pour out their contents. Though the vascular atony may in such states have originated in congestion, yet as this is removed by the effusion while the atony remains, they come at last to be rather instances of passive flux, or dropsy. Chronic ascites, in which the smaller branches of the portal vein may be so obstructed that no fluid can pass through them, presents an exquisite instance of effusion depending upon venous obstruction. The fluid effused in the peritoneal sac varies a good deal, chiefly as to the relative proportions of water and albumen which it contains. In the table subjoined at page 116, one instance is seen in which the water amounts to 988 per 1000, and the albumen to only 0.9, while in another the water does not exceed 704, and the albumen is in the prodigious quantity of 290. Not only does a more or less watery serum exude, but fibrin not unfrequently accompanies it. Large flaky masses of fibrinous coagula are not uncommonly found in the peritoneal cavity after death in cases of ascites, and the same are also seen occasionally in the fluid evacuated by paracentesis. We have also seen blood-globules so uniformly dispersed through the fluid, that there could be no doubt that they had escaped from the congested sub-serous capillaries, and were not accidentally mingled with the effusion. The same products of congestion also occur in the urine when obstructive disease of the heart throws back the blood on the veins. The secretion is albuminous, contains fibrinous casts of the tubes and blood-globules. Decided hemorrhage may also occur, as the result of extreme congestion, which may be dependent either upon venous obstruction or upon an atonic state of the vessels. Melæna, or hemorrhage from the bowels, is an instance of the first, passive menorrhagia and epistaxis, of the latter.

We shall return to the consideration of effusions proceeding from congestion, when we speak of the results of hyperæmia generally. It is a remarkable and instructive fact, that congestion of parts never seems to occasion hypertrophy; or, if this should appear to have taken place, closer examination proves that rather the reverse is the case; that the hypertrophy is what Rokitansky calls *unreal*. Thus, a large bacony spleen or liver may appear to be hypertrophied; but the in-

crease in size is not due to the formation of fresh natural tissue, but to the infiltration and addition of an unnatural product, among which the real structure is found atrophied. In these particular instances the deposits may not have proceeded from congestion; but they afford an exact illustration of what often takes place in congestion in a less degree. It seems also very doubtful whether simple passive hyperæmia, *as such*, does produce even these unreal hypertrophies, or whether in all such instances the crasis of the blood is not also altered in some special manner. After very numerous examinations of congested livers from persons dying with heart disease, we have not been able to convince ourselves that the hyperæmia gives rise to any new product, or that it is an exciting cause of the cirrhotic alteration which may also exist. We have already pointed out the impairment of vital power which congestion occasions, and the causes inducing it, which seem to afford an adequate explanation of the non-tendency to growth and development which is observed both in the affected part itself and in its interstitial effusions. The following sentence from Mr. Simon's lecture recognizes and similarly explains the same fact: "It is true, that much blood is contained in the affected tissue; but it is blood that has insufficient means of renewing itself; and from its long detention in the part it acquires, in an extreme degree, the character of venous blood. Thus, as regards mere bulk of blood, the part is over-supplied, but, in respect of the quality of blood, it may be said to suffer what is equivalent to anæmia: accordingly, the elements of its texture fall into a state of atrophic softening, which terminates in the formation of an ulcer. I think it not improbable that the same fact may contribute to explain the continued non-development of those effusions which arise from passive hyperæmia." It thus appears that atrophy, rather than hypertrophy, is likely to be the result of abiding congestion of any part.

The *effects* of congestion of a part, especially if it be one of some magnitude, may not be confined to the part itself, but may affect the system generally. This appears in two respects, one being a degree of faintness and depression, occasioned by the withdrawal of a considerable quantity of blood, from active circulation; the other, an injurious influence exercised on the whole blood mass by the deteriorated portion, which slowly and partially returns into it again from the seat of congestion. It is clear that an excess of blood poured into one part, and detained there, must leave others imperfectly supplied; the local hyperæmia, according to its extent, produces a degree of general anæmia. That blood which has long been stagnant in a part must be in an unhealthy state is very comprehensible, and that, by its mingling with the general mass of blood, deterioration of the same will be induced, and therewith a cachectic state. Thus, when a congested state of liver exists, there are, frequently, abundant lithates present in the urine; the blood, returning from the liver, conveys into the circulation matters which induce an unnatural state of the renal secretion and more or less of general disorder.

ACTIVE HYPERÆMIA.—DETERMINATION OF BLOOD.

This is the second variety of local hyperæmia that we have to consider. The general phenomena of this condition are manifestly different from those of passive hyperæmia, and convey the idea of increased activity and vigor in the vital process. The flow of blood to the part is increased; the capillaries, without being greatly distended, are well filled, and give a more or less suffused red blush to the face, very different from the duller tint of congestion. In consequence of this filling of the capillary plexus, and, perhaps, also of some slightly increased blastemal exudation, the turgescence of the part is increased, and its temperature elevated. The arteries leading to the seat of active hyperæmia often pulsate with more than ordinary force, so as to have given rise to the idea and term of "increased vascular action." They must also be enlarged to admit the greater quantity of blood that the part receives, and this enlargement becomes permanent; that is to say, the whole vessel assumes larger dimensions, when the hyperæmia is a healthy and natural state. The veins also enlarge, but are not distended as they are in congestion. The sensibility of the part is commonly increased; its function may be, or may not, and the same is true of its growth.

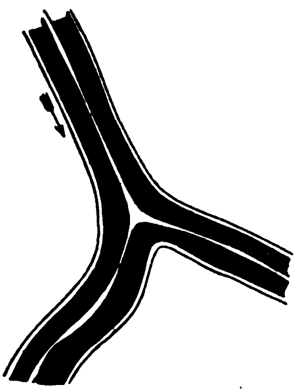
This brings us to the inquiry, whether active hyperæmia may not be distinguished into two forms, one to be regarded as healthy, associated with increased vital power and capacity for action; the other morbid, attending upon and promoting unnatural action, resulting in disease and decay. There are many well-known and oft-quoted examples of healthy hyperæmia, such as the female breasts during gestation and lactation, the uterus during the period of pregnancy, the gums during dentition, the mucous membrane of the stomach while the secretion of gastric juice is going on, and, generally, it may be said, every organ during the time of increased activity and employment. Now, we think it may be affirmed, that, in the above instances, *the most important and characteristic phenomenon is the increased functional energy and vigor* of the hyperæmic part; this we believe to be the main and essential circumstance of which the increased blood-flow is a sequel. Nay, there are many instances, especially among secretory organs, in which the great increase of the product proves that a corresponding increase of the supply of blood must have taken place; a true hyperæmia, in one sense, exists, but it is not apparent, because of the active transformation which is going on. How marked is the difference between this condition and congestion! In the one, functional activity and molecular change at its height, with vascular injection more or less considerable, but not varying in direct, rather in inverse, ratio to it; in the other, vascular injection extreme, while the functional activity is extremely depressed. Now, in proportion as active hyperæmia departs from the physiological condition and becomes morbid—that is to say, approaches towards inflammation—in the same degree does the vital energy and activity of the part appear to be lowered, and the hyperæmia becomes the more marked phenomenon. Thus, a diuretic drug shall be administered to two individuals: in the one, the flow of urine

shall be considerably augmented, the vital power of the kidney predominating over the hyperæmia excited; in the other, the flow of urine shall be diminished, and the secretion become bloody and albuminous, evidencing the predominance of the hyperæmia over the vital power. So, too, in fever. The skin, at one time, shall be dry, and burning, and red, with vascular injection, but its vital power of secretion and exhalation is in abeyance. But a change comes, the tissue regains its functions, and pours out a healthy moisture on the surface; and now the hyperæmia, though it may continue in some degree, is no longer predominant. How different must be the condition of the gastric mucous membrane in the hyperæmia excited by a few grains of ginger, and that produced by a few grains of arsenic! No doubt the one form of active hyperæmia may pass into the other. The quantity of blood which a healthy tissue was able to employ, and which was requisite for the unusually vigorous discharge of its function, may become too much for the same tissue when debilitated by over-use. A brain ministering to an active mind, requires and receives a greater supply of blood than that of the waterman "who rows along thinking of nothing at all." So long as the cerebral energy is not overtasked, the hyperæmia will tend to no injurious result, but will only supply the necessary pabulum for the material changes connected with thought. When, however, the time arrives that the delicate organ needs repose, then if the strain be continued, and the hyperæmia kept up, it is manifest that a morbid state will soon supervene, in which the hyperæmia may yet further increase, and the natural energy be still more diminished, till, together with symptoms of disordered and erring action, inflammation, or some other result of hyperæmia, occurs. The phenomena we have alluded to are of every-day occurrence. No doubt can exist about their reality, though different opinions may be entertained as to how they should be interpreted. We shall immediately proceed to consider more closely the different views that have been advanced, but we would ask especial attention to this point which we have dwelt on, viz: that in one form of active hyperæmia, the vigor of the tissues for vital action is increased, while, in the other, it is diminished. In the one the hyperæmia supplies a want, in the other imposes a burden.

Now, before we enter on the consideration of the mode in which active hyperæmia is brought about, let us refer shortly to one or two physiological points, which must form the very basis of all our attempts at explanation of the phenomena. All tissues may be regarded as consisting of vessels, nerves, and the proper elements of the tissue. The vessels, it is true, may be more or less closely woven up with the tissue, or even may not actually penetrate it, but still they are essential. The nerves convey an influence which may affect the bloodvessels or the tissue. The elements of the tissue, be they cells, or originally derived from cells, have certain special endowments, which, when called into action, increase the flow of blood to the part; at least, this seems only another form of putting the undoubted fact—that exercise of a part causes more blood to flow thither. The bloodvessels consist of arteries, capillaries, and veins. The arteries have a truly contractile coat, which, under some kinds of stimulation, may even produce obliteration

of the channel. This contractile coat resembles, but is not quite identical with, organic muscular fibre. It possesses, so far as we know, no other property than that of contracting. There is not the least reason to suppose that it has any power of active dilatation. The capillaries have a simple, homogeneous, membranous well, in contact with the tissue proper of the parts. It does not appear that this possesses any contractility. The contractility of the veins has generally been considered much more doubtful than that of the arteries. Weber did not find them respond to the stimulus of cold as the arteries did. Mr. Wharton Jones, admitting that they do undergo some variation in size, in virtue of the contractility of their outer coat, still expresses himself clearly, "that constriction or dilatation of veins cannot be observed actually taking place, as it may be in the case of the arteries;" and "secondly,

Fig. 9.



Contracted artery, from Wharton Jones's Essay.

that the degree of constriction or dilatation which the veins in any case present, is very small in comparison with that which the arteries undergo." Professor Paget's observation seems conclusive as to the contraction of both veins and arteries under a mechanical stimulus. He says, "if, as one is watching the movement of blood in a companion-artery and vein, the point of a fine needle be drawn across them three or four times, without apparently injuring them, or the membrane over them, they will both presently gradually contract and close; then after holding themselves in the contracted state for a few minutes, they will begin again to open, and, gradually dilating, will acquire a larger size than they had before the stimulus was applied." It must be noticed, that Mr. Paget's observation has reference to the wing of the bat, while Mr. Wharton Jones's conclusion has been formed from examination of the web of the frog. The former, as a warm-blooded mammal, is no doubt a better representative of man than the reptile.

What we can see, by the microscope, in parts that are the seat of active hyperæmia, in consequence of the application of a stimulus, is by the very accordant testimony of the best observers as follows: When a moderate irritant, such as tincture of capsicum, or a drop of some

essential oil, is applied to a transparent part, the arteries speedily dilate, and a rapid flow of blood through them ensues. This seems to tell on the capillaries and veins, which become dilated also, so that vessels, which before scarcely admitted blood-globules, are now traversed by great numbers. Sometimes the dilatation seems to be preceded by constriction of the artery; but this does not constantly occur, and when it does, is of brief duration. It has been often said, that the flow of blood was *accelerated* in arteries that were contracted; but the reverse seems rather to be the truth. It is the *dilatation* of an artery that causes the current to become rapid, doubtless in consequence of the less resistance opposed to the *vis à tergo* of the heart. Professor Paget writes: "As the vessels are contracting, the blood flows in them more slowly, or begins to oscillate; nay, sometimes, even before the vessels begin visibly to contract, one may observe that the blood moves more slowly in them, as if this were the first effect of the stimulus. Nor am I sure that I have ever seen (what is commonly described) the acceleration of the flow of blood in the contracting vessels. Such an acceleration, however, is manifest, as the vessels reopen; and as they dilate, so, apparently in the same proportion, does the flow of blood through them become more free, till at length it is quite manifest that they are traversed by both fuller and more rapid streams than passed through them before the stimulus was applied." Mr. Wharton Jones's observation is to the same effect: "In one case," he says, "the arteries of the web were more or less constricted, the circulation sluggish, the blood in the capillaries here and there stagnant. A drop of the solution of sulphate of copper with vin. opii was applied, whereupon the arteries immediately became dilated, and the circulation brisk." Dr. Williams, in his work, so often referred to, had previously maintained and clearly illustrated the same view. It may therefore be considered established, that in active hyperæmia one principal feature is dilatation of the arteries; while in passive hyperæmia, the veins and the capillaries opening into them are dilated, and the arteries either are not enlarged, or are constricted. Now, it may very naturally be asked, in what way does the stimulus applied bring about dilatation of the arteries? All that we know of the habits of contractile tissues, leads us to believe that they can only respond to a stimulus by exerting their contracting power, and there is no known instance of active elongation. How is it, then, to be conceived that a stimulant, applied locally, which does not affect the action of the heart, can cause dilatation of the bloodvessels? Henle conceives that the stimulus, acting on the sensory nerves of the part, excites in them a state, which, being communicated to the spinal centre, is reflected on the vascular nerves, occasioning them to become paralyzed, and therewith the contractile coat of the vessels also. This theory (the neuro-pathological), though it has found credit with many, really seems only to shift the difficulty from the bloodvessels to the nerves. It is just as contrary to experience that excitation of a sensory nerve should paralyze a motor, as that the stimulation of a contractile tissue should make it elongate. Stilling's modification of the Neuro-pathological Theory, rests on the assumption that there is a continual reflected influence from the sensory upon the vas-

cular nerves, so that when the sensory are paralyzed, the vascular are paralyzed too, and when the former are excited, the latter are also; but if, as we know, a paralyzed limb can be the seat of active hyperæmia, and if, as Mr. Simon has shown, "the absence of a spinal cord, or the division of all the roots of the nerves, or the section of the lumbar and sciatic plexus, will make little or no difference as to the certainty with which an irritant, applied to the web of a frog's foot, will quicken the circulation there, and subsequently lead to its retardation and arrest," it is abundantly clear that all such views are quite inadequate to account for the phenomenon in question. It is to be remembered, that contraction and dilatation of vessels, and increased and more rapid flow of blood through them, are things that, by the aid of the microscope, we can see; but the molecular movements of nutrition and secretion, which we believe to influence and modify the circulation through a part, we cannot see; they are as real and as potent, but they are, except in their results, invisible. Feeling, however, the importance of ascertaining every cause that we suppose to be concerned in the production of a phenomenon, to be a "causa vera," we briefly put together the arguments which appear to us conclusive, that what we have called the "nutrition force," and Dr. Carpenter the "capillary," does really exist, and is concerned in producing the state of active hyperæmia. (1.) When a part is not employed (a limb, for instance) for some time, it wastes and atrophies. Its bloodvessels become smaller, and its temperature falls. Manifestly, the circulation of blood through it is diminished. (2.) When a part (as a limb) is actively employed, it enlarges, its temperature is increased, its bloodvessels are more developed, and the quantity of blood passing through it is evidently greater. (3.) When a gland is excited to increased action, as the mamma of the female, the flow of blood to it is increased, and the vessels become enlarged. (4.) In plants it has been observed, among other instances of the influence of local stimuli, that a branch of a tree, growing in the open air, which is brought into the atmosphere of a hothouse, will vegetate during the winter, and draw up sap through the stems and roots, while the other branches remain in their ordinary state. (5.) In many of the lower invertebrata, the movement of the nutrient fluid seems to be evidently independent of the action of a heart on the vessels. (6.) Dr. Houston's case of an acardiac fœtus has proved, in the judgment of those most competent to decide,[1] "that a fœtus may grow to a considerable size, and have its various tissues well developed, without any connection with the twin fœtus, by means exclusively of a circulation of its own, of which a heart forms no portion, or upon which it can exercise but a very remote influence." (7.) Though the phenomenon of blushing, and some other local determinations of blood, may be accounted for by an alteration taking place in the caliber of the bloodvessels, their channels being widened, and more blood admitted, yet that of hyperæmia, excited by a local stimulus, appears to us quite impossible to explain in such a way. We would refer more particularly to the interesting experiment, recorded by Mr. Simon (p. 96 of his

[1] Todd and Bowman, Phys. Anat. vol. ii. p. 372.

Lectures on Pathology), in which hyperæmia was induced by the local application of a stimulus to a part, which had lost all trace of sensibility. This appears to us to afford conclusive proof, that neither the action of the heart, nor that of the bloodvessels, but only the nutritive force, heightened by the action of the stimulus, could have produced the local erythema. This justly eminent authority adds: "Altogether we may, I think, take it as an established certainty, that the first change which occurs in an inflamed or overgrowing part, and which leads to its becoming loaded with blood, is not a reflex change operated through the nerves, but is a direct change, operated by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood." He compares it to "a vortex, established in the place of the irritant, causing all the adjoining streamlets of blood to converge in swifter channels towards it." Professor Paget says: "I think I can be quite sure that the velocity of the stream, in any vessel of an inflamed part, is not determined by the diminution or enlargement of the channel. Without change of size, the stream may be seen decreasing from extreme velocity to complete stagnation. On what the alteration of movement of the blood in such a case depends, I cannot tell; but we have facts enough to justify such an hypothesis as that there may be some mutual relation between the blood and its vessels, on the parts around them, which, being natural, permits the most easy transit of the blood, but, being disturbed, increases the hinderances to its passage."

The foregoing arguments and authorities must be allowed, though in opposition to Dr. Williams and Rokitansky, to carry very considerable weight with them; and the remark will appear justified, that it really seems far too exclusive and one-sided a view to consider only the blood and the vessels as the agents concerned in hyperæmia, the common initiatory step of inflammation, and to deny to the essential elements of the part any share in the production of a state by which they are so importantly affected. We therefore recognize an increased attraction of the blood towards the part which is stimulated, as one cause of active hyperæmia, and the principal, and we regard the dilatation of the arteries as a secondary, but not unimportant. But the influence which the tissues exert on the circulation, in virtue of their "nutritive power," we may be sure is not only an attraction which may be increased or diminished, but also an alteration which the attracted blood undergoes, and, having undergone, is either repelled or pushed on by the advancing current. We may illustrate this motive influence by the example of light bodies, when acted on by electricity. Two pith balls, one of which is in a negative, and the other in a positive state, will attract each other strongly; but as soon as they both become negative or positive, they forcibly repel each other. Some similar relation must subsist between the blood and the tissues. The arterial blood is heterogeneous to the tissue, and is attracted to it. Having become venous, it is no longer so, and it ceases to be attracted, perhaps even is repelled. Now, we may conceive the attractive force to persist, or even to be exalted, while the change impressed in nutrition may be greatly lessened. Blood will then accumulate in the part, from not having undergone that

vital change which it should, and the part will be hyperæmic. This would be the case in active hyperæmia of a morbid kind—in that which forms the first stage of common inflammation, in which the vital endowments of the part are lowered, and its functional activity lessened. In healthy hyperæmia, on the other hand, the attractive and the changing influences are both increased. The blood does not accumulate, but only ministers adequately to the increased functional activity of the part. We are anxious to avoid, as far as possible, speculating beyond the limits of actual observation, but we would ask, whether some such interpretation as we have offered be not necessary to explain the different event in two cases of suckling females, one of whom has the child put to the breast early, and by the mental and psychical influence brought to bear, has the functional activity of the gland aroused, so that the hyperæmia as it arises is converted into a copious flow of healthy milk; while the other, who has the child kept from her for two days, and whose mammæ are left unaroused and unstimulated, suffers from overwhelming hyperæmia, which issues in inflammation and suppuration.

We now pass from the consideration of the mode in which active hyperæmia is induced, to that of its effects. These, as already intimated, are different in the healthy and morbid varieties. In the first, the growth of the part, if the hyperæmia continue long enough, is increased; it undergoes a true hypertrophy. Of this we have the best examples in the muscular tissue. At the same time, the function is more vigorously exercised; it has more capability for, and it performs more work. Of this the brain, under the influence of moderate determination of blood—such as some of our great orators used to induce by pretty free libations of wine—is a good example. Similar instances among glandular organs we have already noticed. That of the ovaries and uterus, at the catamenial periods, is very remarkable, and is evidently connected with the reproductive nisus, which manifests itself especially at these epochs. There seems no ground whatever to regard it as originated by nervous influence, but rather as the result of a mode of growth and nutrient action peculiar to these organs. The discharges of the ovarian ovum, and the catamenial flow, are the results of this hyperæmia, but it is itself excited by the spontaneous activity of the structures. Thus curiously, as we see also in many other instances, are linked together the increased action of an organ, and the increase of its supply of blood; in the healthy state, the former usually takes the initiative, and produces the latter, but is itself reacted on by it, increased, and carried on.

But it is rather with morbid hyperæmia that we are concerned as pathologists. This is, in a very great number of instances, the commencement of inflammation; but we shall not speak of inflammation as one of its results, but consider it separately. The effects of morbid hyperæmia are generally unnatural excitement, or oppression of an organ. The part contains more blood than it is able to manage, its healthy play is interfered with, and it is either goaded into a false, aimless, and exhausting activity, or it is actually oppressed and enfeebled directly. The chief features of the condition are, probably, increased attraction of blood to the part, with diminished vital change, and undue dilatation of the arteries leading to it.

If the brain be the seat of determination of blood, in a morbid sense, there will be throbbing of the carotids and their superficial branches, restlessness, more or less intolerance of light and sound, diminution of the power of attention and application, dreamy and disturbed sleep, irritability of temper, attacks of giddiness, &c. The face and eyes are apt to be flushed, and the feet cold. The uneasy sensations about the head are increased by stooping, or the recumbent posture. The kidney, in cases of acute anasarca, manifests an excellent example of morbid hyperæmia. It is enlarged and turgid with blood, but its texture is not apparently altered. Its secretion is scanty, loaded with albumen, and with fibrinous concretions, and epithelium of the tubes. No doubt can exist that its functional energy is gravely impaired. In active menorrhagia we have a third instance in which, from various causes, a morbid hyperæmia of a hollow organ lined by a secreting mucous membrane is induced, the results being pain and uneasiness in the region of the affected part, increased sensation of heat, tension and throbbing, which are relieved by the discharge of a fluid more completely sanguineous than the natural secretion; in fact, by an almost real hemorrhage. In such an instance, it is not only the mucous lining of the uterus that becomes hyperæmic, but the whole organ, with its thick muscular walls. Their tissue is loosened up and swollen by the quantity of blood admitted, so that the size of the organ is increased; and if this hyperæmia should not in great degree subside, the result may be a permanent enlargement and congested state of the uterus. The foregoing examples, taken from different organs, will serve as sufficient illustrations of the effects of active hyperæmia. It seems, however, desirable to allude, somewhat more in detail than has yet been done, to *hemorrhage*, *flux*, and *dropsy*, considered as results of hyperæmia in general.

These may be regarded as the effusions of hyperæmia, as distinguished from inflammation. No doubt they do also occur in cases where inflammatory action is proceeding; but still they are not the special and characteristic products of this state.

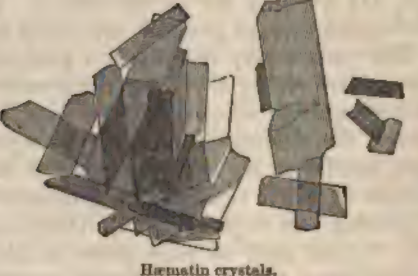
Hemorrhage implies the effusion of blood in mass, not merely of some of its constituents; exudations, therefore, which are only colored by hæmatin, do not constitute hemorrhage. The best character of an hemorrhagic effusion is the presence of large masses of blood-globules imbedded in fibrinous coagula. Such may be found either from an opening in a vessel of some magnitude, or from numerous capillaries. In every case where blood is effused in any quantity, the walls of the vessels must have given way; and perhaps this is the case in every instance where a blood-globule escapes from its channel, though it is not, to our minds, absolutely certain that there is no such thing as the hemorrhage by exhalation of the older writers. Hemorrhage may take place either in solid parenchymatous organs, or in those that inclose cavities and form canals. In the first case, the substance of the organ undergoes more injury than in the latter. An effusion of blood into the brain is a most serious thing; on the surface of the Schneiderian membrane it is a mere trifle. When a large quantity of blood is suddenly extravasated in a solid organ, it ploughs up and disorganizes the

tissue, and forms therein a cavity for itself, where it lies like a mass of black currant jelly: the walls of the cavity are usually ragged, and soon become stained to some depth by altered coloring matter. But the extravasation may occur in a very different manner, affecting a great number of points at once, and having the appearance of a multitude of red dots scattered about, or of minute streaks. This is termed capillary apoplexy, to distinguish it from the other form ("apoplect. herd" of Rokitansky). It is often seen in the gray matter of the cerebral hemispheres after death, from concussion of the brain.

If the hemorrhage, though taking place in the same way, be more abundant, the spots and streaks approach closer together, the tissue becomes more swollen, and, at last, may become thoroughly red, the blood having penetrated completely between and among the elements of the parts. Rokitansky enumerates the following exciting causes of hemorrhage: (1.) Hyperæmia of very great intensity, of whatever kind. This may occur in various conditions of the system, but of course the local strain will be most severe when a state of plethora exists at the same time, and the action of the heart is powerful. We have seen considerable hæmoptysis take place in a healthy person after very severe exertion. This might be considered as an instance of hemorrhage from intense active hyperæmia. The large evacuations of blood that are poured out from hæmorrhoids, exemplify the same result from passive hyperæmia, as also do cases of passive menorrhagia. (2.) The hyperæmia that takes place in some kinds of inflammation which have been hence named hemorrhagic. This tendency is materially promoted by the delicacy and laxity of the tissue affected. The lungs are scarce ever inflamed without some amount of hemorrhagic exudation taking place, viz: the rusty sputa of pneumonia. Where scurvy is prevalent, hemorrhage seems more prone to accompany inflammations, as might, indeed, be expected. A form of pericarditis has recently been described by Dr. Kyber, which he terms "pericarditis scorbutica," in which, after death, large quantities of bloody coagula, together with effusions of lymph, are found in the serous cavity. Hemorrhage from this cause is apt to attack morbid growths, especially the softer varieties of cancer. (3.) The vessels may rupture and pour out blood in consequence of the tissue surrounding them becoming more spongy and lax, so that they are not adequately supported. This appears to be the cause of hemorrhage from the decrepit uterus of the aged. (4.) Textural disease of an organ, rendering it more brittle or soft than natural. The most marked instance of this is the fatty, degenerated heart, which has often undergone spontaneous rupture. (5.) Alterations of consistence of the coats of the vessels, such as take place in atheromatous disease or in chronic inflammation; this may affect the smallest vessels or the largest trunks, but is almost confined to the arteries, and may be accompanied with dilatation (aneurismal) or not. (6.) Ulceration of the surrounding tissues may give rise to hemorrhage, by involving some of the vessels. Severe, or even fatal, hæmatemesis, from ulcers of the walls of the stomach, is not uncommon. The hyperæmia which issues in hemorrhage, may be occasioned by obstruction to the venous current, as in hæmorrhoids from diseased liver, and pulmonary apoplexy from

obstructive disease of the heart; or it may be caused by intense irritation, as when bloody discharges are occasioned by drastic purgatives, or hæmaturia by stimulant diuretics. Posture may prove the cause of hemorrhage when the tonicity of the vessels is very low. Thus, stooping has been known to occasion cerebral, and the erect posture uterine, hemorrhage. The influence of malaria and cold has been before noticed. There seems to be a kind of hemorrhagic diathesis; at least, Dr. Copland states that hemorrhages are more common in the offspring of parents who have suffered from them than in others, and that the tendency is observed in several members of the same family. Hemorrhage from the rectum, urinary organs, and uterus, is said by Chomel to occur oftener in cold than in warm seasons, and epistaxis and hæmoptysis to be more frequent in summer than in winter. Dr. Prout observed a peculiar tendency to renal hemorrhage during the time that cholera was prevalent. Age seems to have an influence in determining the seat of hemorrhage. Epistaxis is most common in children, hæmoptysis in the early period of life, and hæmorrhoidal discharge in the aged. The blood, when effused, may remain in a liquid state for some time, or quickly coagulate. When it is poured out into the substance of a part, it undergoes, after a time, the changes which are commonly observed when a superficial part has been bruised. These consist in alteration of the color of the hæmatin, which passes "from a dark red into a blue, then into a brown, and lastly, into a yellow color, before it entirely disappears." At the same time the blood-globules, at least in many cases, undergo peculiar changes; they become massed together, and sometimes included in a kind of cellular envelop; they waste and shrink up, until there remain at last only minute yellow or orange-red granules, which evidently consist chiefly of pigment. Such are not unfrequently found in the straight tubuli of kidneys affected with M. Brightii; they are the undoubted records of former hemorrhage. In some cases the

Fig. 10.



Hæmatin crystals.

altered hæmatin takes the form of crystalloid, elongated, rectangular tablets, which vary very much in size, and are colored more or less deeply by red matter. The formation of these seems to be promoted by the addition of water. They were extremely well seen in a case which we witnessed of cystic disease of the kidney, in which several large dark clots were contained in a cyst of extraordinary magnitude. The

fibrin, and other residue of the extravasation, together with broken-up fragments of the tissue, are gradually reabsorbed, the solid substances undergoing liquefaction, chiefly in the way of fatty transformation. A further change may take place, not so much in the effused blood, as in the parts around it. These, which are at first ragged and torn, undergo more or less inflammation, which ends in the effusion of a solidifying blastema; this fibrillates, and passes into the state of more or less perfect fibrous or areolar tissue, and thus forms a capsule or cyst, inclosing the now more or less altered blood. Rokitansky describes the cyst as being lined by a colored, soft, gelatinous, loosely-adherent layer, formed from the coagulum, which, at a later period, by fibrillating, and even developing vessels, assumes very much the aspect of a delicate serous membrane. The contents of the cyst may be a gelatinous or serous fluid alone, or with more or less traces of a vascularized areolar tissue. In some cases absorption takes place completely, and the cavity is obliterated by the adhesion of the opposite sides, and the formation of a linear cicatrix. This, however, is not the most frequent issue, in consequence of the following impediments: (1) a large size of the cyst; (2) retraction of the surrounding tissue, depending partly on its atrophy, partly on its induration; (3) the deposition of the fibrin, either as a central lumpy mass, or as a thickish, peripheral, capsulating layer. The effused blood-mass may undergo a different kind of change, in consequence of absorption of its watery parts, and become, in this way, a kind of tumor, termed an hæmatoma. Dr. Walshe classes this along with other growths, but we think it better to consider it as a simple result of hemorrhage, and this for three reasons: (1) that it presents no higher structural character than that of fibrin; (2) that it is generally devoid of vessels; (3) that it does not appear to increase by growth in the proper sense of the term. Dr. Walshe describes an hæmatoma from the spinal meninges, which had been produced by a blow, as of the size of a walnut, of pale straw color, and of fine granular texture when closely inspected. "Such tumors exhibit, microscopically, the qualities of fibrin; fibrils gelatinizing with acetic acid, amorphous fragments, granules, and molecules." "Their chemical relations are those of fibrin." An epithelial investment covers the surface, and makes it smooth, but there is usually no enveloping cyst. Hæmatomata occur in serous and synovial cavities, beneath fibrous and mucous membranes, in parenchymatous organs, in muscular masses of the limbs, in the substance of certain new products, especially encephaloid cancers, in cavities accidentally formed in the tissues, as in tuberculous cavities of the lungs. An hæmatoma thus formed, and being essentially a fibrinous mass, may undergo certain other changes; saline earthy matter may be deposited in it, inducing a state which is more correctly named *cretification*, than ossification; melanic pigment may probably also form in it. It may, perhaps, undergo a development to the somewhat higher stage of fibrous tumor, and sometimes even true bone may be formed within it. A vascular plexus has been observed in several instances in tumors of this kind; and though it may be objected that this has been developed in superadded exudation-matter, yet it appears to us very much more probable that the persistent fibrin afforded the developmental nidus from its own substance. One result of hemor-

rhage, even in lesser degrees, may be the persistent discoloration of the tissues from the presence of brown or black pigment, which is diffused among the elementary parts in a finely divided condition, as one of the transformations of the effused hæmatine. Hemorrhage, like the hyperæmia from which it results, may be active or passive, sthenic or asthenic. The former variety is associated with the same general condition of the system which characterizes active hyperæmia, the latter, in the same way, is connected with passive hyperæmia. Rokitansky says of the condition occasionally observed, in which hemorrhage takes place to an alarming amount, even from a slight cause, which he terms Hæmorrhophily, that it depends, as far as we know at present, on an unusually delicate construction and vulnerability of the vascular membrane, together with a thin, watery quality of the blood in general.

FLUX AND DROPSY.

We have already in part noticed these results of hyperæmia, more particularly of the passive variety, but it seems desirable on account of their great importance and frequency to review them separately. The term flux may be properly applied to a discharge of various kinds taking place from a mucous surface, or from a glandular organ connected therewith; the term dropsy to an effusion of fluid in serous or synovial cavities, or in the areolar tissue. Fluxes will be active or passive according to the kind of hyperæmia which occasions them; the same can scarcely be said of dropsies; the very great majority of them are passive. It is necessary to fix some limitation to the kind of fluid that may be said to constitute flux or dropsy, as there are many exudations which require to be distinguished on account of their different nature. A sero-purulent effusion, on the secretion of a serous cyst, would not come under the present head. Perhaps we shall be nearly correct if we say that a fluid similar to, but more aqueous than the liquor sanguinis, mingled in the case of flux with a varying quantity of mucous secretion or desquamated epithelium, mature or immature, and if proceeding from a gland mingled with more or less of its secretion, is that which properly belongs to this kind of morbid action.

Of active fluxes we have a good example in miniature in a common sneeze; the peculiar sensation demanding the reflex expiratory contraction is no doubt occasioned by the turgid state of the bloodvessels of the Schneiderian membrane pressing upon the interwoven nerves; this hyperæmia quickly terminates in a muco-serous effusion which the blast expels, and after one or two such acts, all is quiet again. More considerable, and much more enduring is the hyperæmia in the state of coryza, and the serous flux is of course much more prolonged. In this instance we have an opportunity of observing a quality of the fluid of a serous flux which is very common, viz: that it is especially acrid and irritating, so that it will sometimes excoriate the parts over which it flows. What gives it this quality is not very apparent; it seems at least doubtful whether it is merely an excess of the natural saline ingredients of the blood. We should rather suppose it to be some organic acid salt

of new formation. The fluid under the microscope exhibits very little trace of corpuscles, and is alkaline.

Choleric diarrhœa is the extremest example of active morbid flux, the whole blood seems to rush to the intestinal surface, and pour out its fluid part, minus the greater part of the albumen and fibrin. The gruel-like evacuations consist of water and saline matter, with some trace of albumen in solution, and a large quantity of columnar epithelium.[1] The reaction of the fluid is alkaline. The filtering action of the intestinal membrane in this instance is very marked, and well worth noticing; it is, indeed, extraordinary, that, while so rapid a rush of blood is going on to the exhaling surface, the effused fluid should be so considerably altered from that which arrives thither; one would have expected it to contain at least as much albumen as the passive exudation of ascites. We would ask whether the case of choleric diarrhœa, as well as the similar condition from drastic purgatives, do not absolutely prove the existence of a power influencing the circulation other than the *vis à tergo* of the heart with the regulating contractility of the bloodvessels? Does it not also demonstrate that it is this nutrition force of the tissues, as we have called it, which determines whether an hyperæmia shall issue in a flux or in an inflammation? In profuse salivation arising from the administration of mercury, or other causes, we have a good instance of an active flux, from a glandular organ, although the fluid is in this case almost identical with the natural secretion. It is not unfrequently observed, that if an active flux be suddenly checked, it will be transferred to some other part, where it may, perhaps, produce much more serious consequences. The part to which the metastasis takes place may be a solid organ, or a secreting surface; in the first case dangerous hyperæmia, and possibly hemorrhage, may occur; in the second, the hyperæmia will relieve itself by a free effusion of fluid. Thus arrest of the menstrual discharge by cold is often followed by determination of blood to the head, arrest of an habitual diarrhœa by the same result, or by the supervention of ascites. The characteristics generally of active fluxes are those of active hyperæmia, which is more or less apparent according to the amount of the effused fluid; if this be considerable, the hyperæmia is dissipated as fast as it arises. Active dropsies are often termed acute, or febrile, and are not always easily distinguished from inflammatory effusions. An almost certain means of distinction is, to observe whether the effused fluid is even slightly turbid with flakes of lymph, or puriform corpuscles; the presence of these is decisive of the inflammatory nature, or at least of some degree of coexisting inflammation. Acute anasarca affords one of the best instances of active dropsy, the interruption of the action of the kidneys, at the same time that it deteriorates the quality of the blood, and renders it less fit to circulate in the vessels, diminishes considerably the separation of fluid from it, so that from both these causes there arises a tendency to the effusion of fluid in the areolar tissue, or in other parts. The tenseness and firmness

[1] In one case we examined, the gruel-like fluid contained feebly formed nuclear particles, with a few granular globules and cells, and an abundance of granular matter; there was little or no epithelium in the evacuations, and some was found *in situ* 38 hours after death.

of the anasarcous swelling in many of these cases lead to the belief which direct observation has confirmed, that the effused fluid contains some amount of fibrin, which coagulates among the elementary parts of the tissue, and makes them more dense and stiff. The same thing occurs also in dropsies of serous cavities, and has been particularly noticed by Vogel, under the name of *Hydrops Fibrinosus.*

Vogel does not seem to discriminate between fibrinous dropsy, resulting from hyperæmia, and that resulting from unequivocal inflammation; and probably it is not necessary. The one condition is the inceptive of the other, and passes into it by imperceptible grades, or may exist in various degrees along with it. In fact, in this instance, and all through our study of pathological anatomy, we cannot too constantly bear in mind that, though we take, and are justified in taking, for description, certain typical forms of living and acting, yet these are seldom rigidly defined, but pass easily into each other, or coexist in the same part variously combined. The simple unmixed case is the rare one, the multiple and the complex the common. Having premised thus that fibrinous dropsy may be, and often is, the result of an inflammatory process, as well as of an active hyperæmia; but regarding it as more properly the product of the latter than of the former, we proceed to follow the account given of it by the excellent pathologist just quoted. He remarks that "it may occur either in serous cavities (as in the pleura, arachnoid, peritoneum, or pericardium), or may collect in the parenchyma of organs." In both cases, however, it is essentially similar. "Examined immediately on its discharge, the fluid resembles in all points that of serous dropsy. This either is perfectly clear and colorless, or else more or less turbid, opalescent, and of a greenish, yellow color; and, examined microscopically in its recent condition, exhibits either no solid particles" (the case of pure hyperæmia), "or only such as may be accidentally present, as occasionally minute amorphous coagula of fibrin, pus-corpuscles," &c. (inflammatory complication). "Some time after its discharge, the whole fluid generally coagulates, in consequence of holding fibrin in solution, and forms a homogeneous, tremulous jelly, which, after standing for some time, separates into a partially consistent colorless, or reddish-yellow clot of coagulated fibrin, and a clear yellow fluid, analogous to the serum of the blood." "The coagulated fibrin appears under the microscope as a perfectly amorphous mass, and devoid of any traces of cellular structure." The effused fluid bears a pretty close resemblance to the liquor sanguinis, containing the same saline and animal matters, but a less proportion of albumen and fibrin. Vogel considers that the effusion of fibrinous fluid depends on the transudation taking place through the walls of the capillaries, while a simply serous effusion is poured out through the walls of the veins. We cannot join in this view, as it seems to us impossible that transudation should take place in any case exclusively from the one or from the other. All the capillaries and minute veins in hyperæmia are alike gorged, and there is no sufficient difference in the structure of their coats to make it probable that they affect at all materially the nature of the exudation. The real cause of the difference between fibrinous and serous dropsy we believe to be the different quality of the blood, the existence of a differ-

ent crasis, to use Rokitansky's term; in fact, the presence of a greater amount of fibrin. Vogel recognizes the dependence of fibrinous dropsy on dynamic causes; *i. e.* its association with active hyperæmia, while serous dropsy depends more upon mechanical. This is true; but at the same time we have seen some amount of fibrinous effusion occur in passive dropsy, depending upon mechanical obstruction to the circulation. The fibrin contained in the fluid may either remain for a long time (days, or even weeks) within the body uncoagulated, and coagulate after its discharge, or it may pass at once, while within the system, into the coagulated condition. It may also serve as a blastema for organic formations. This we shall speak of further on, under the head of "Exudations."

Passive fluxes are of extreme frequency, and are almost invariably associated with debility. Their copiousness, the aqueous nature of the fluid, the frequent pallor, and non-elevated temperature of the parts from whence they proceed, as well as relaxed conditions of these, may be said to be their general characters. They take place, as is manifest, from mucous surfaces, or from the glands that open upon them, and are, in consequence, mingled, more or less, with liquor muci, and with epithelial particles, in various stages of formation. In some cases, as in bronchorrhose, occasionally they may depend upon venous obstruction; but their most essential cause seems to be a peculiarly relaxed and toneless state of the walls of the vessels, and of the tissues affected. This is confirmed by the beneficial effect of astringents, locally applied. At the same time, there is no doubt that the state of the blood, and of the system generally, has an influence upon them, and that, as the quality (the crasis) of the former is improved, so the debilitating profluvium will diminish. It is reasonable to suppose that in this way the walls of the vessels are brought into a condition of more healthy tone. The peculiar condition of the tissues, as to their vital endowments, seems to be the only sufficient cause to which we can at all ascribe the different phenomena exhibited by the same tissues, under similar circumstances. One person shall have a chronic bronchitis, with puriform expectoration, while another person is suffering under bronchorrhœa, although the circumstances may be similar, and the two affections very much alike in their outset. When we speak of a toneless and relaxed condition of the vessels being the main cause of the flux, we do not so much mean to imply a defect in their contractile power (though this doubtless exists), but rather such an alteration of their texture, as that they are much more transudable by the aqueous part of the blood than is normally the case. In health, a certain slight exudation takes place from the capillaries, and all the minuter vessels, forming a nutrient atmosphere, in which the tissues are bathed. The exact composition of this is uncertain; but, as the following experiment shows, it probably contains less albumen and fibrin than the liq. sanguinis. Valentin, having made an albuminous solution of a certain specific gravity, placed it on a filter of stretched serous membrane, and, on examining the fluid which passed through, he found its specific gravity reduced; that is to say, some of the albumen was left behind. It is probable, and seems proved by pathological experience, that increase of the pressure upon

the fluid causes it to transude in a less altered state; so that this, whether it be a *vis a tergo*, from increased cardiac impulse, or caused by an obstruction in the onward direction, must be one cause of an effusion containing much albumen and fibrin also. But the common watery and mucous effusions, which constitute such fluxes as those of leucorrhœa and bronchorrhœa, contain little, if any, albumen, and no fibrin, and are chiefly remarkable by the quantity of their aqueous and saline contents. The same may be said also of the intestinal and cutaneous fluxes that take place in phthisis, and in other exhausting diseases.[1] In all these it seems certain that the natural filtering power of the walls of the vessels is changed in such a way that they allow the aqueous and saline part of the blood to transude with extreme rapidity. At the same time, the crasis of the blood itself is altered; it turns, as the popular phrase is, to water; *i. e.* its corpuscles and its organic matters are not formed in due proportion, but, on the contrary, waste and diminish, so that the colliquative discharges from the tissues are promoted, and kept up by the (as it were) deliquescing blood. It is interesting to observe that the filtrating property of the vascular membrane is capable of being influenced through the nervous system. Thus, after a fit of hysteria, a quantity of limpid aqueous urine is passed, much more than would have been voided had no such event occurred. This must depend upon an alteration of the condition of the Malpighian tufts, and perhaps of the capillaries of the tubular venous plexus. We have noticed something of the same kind after a small dose of opium. Almost the only instances of fluxes taking place from the glands, with which we are acquainted, are those which the kidneys afford. That of diabetes depends, as is well known, upon a diuretic substance, sugar, circulating in the blood, and not undergoing the decomposition which it should normally. Diabetes insipidus was believed, until recently, to depend upon some other and different cause; but it has been shown by Thenard and others that the only difference is that the sugar which is present, and produces the diuretic effect, is tasteless. In cases of polydipsia, where an excessive quantity of urine is passed, of low specific gravity, the flux depends simply on the injection of an undue quantity of liquid, in consequence of extreme thirst. Discharges, such as those of chronic bronchitis, or chronic dysentery, which were, in their commencement, of truly inflammatory nature, but afterwards become more of the nature of fluxes, often contain a very large amount of muco-purulent matter, and cause a proportionally severe drain on the system. These, however, do not present the hydræmic condition of the general system, before alluded to.

Passive dropsies are the commonest of all; all the cardiac dropsies, and most of the renal that we meet with, are of this kind. Their very aspect excludes the idea of increased action (however the term may be understood), and naturally suggests that of some obstruction to the circulation, with diminution of the vital energies. The surface is generally pallid, or of a dull venous hue, the animal heat is diminished, the anasarcous swellings pit easily on pressure, the effect of gravity upon them

[1] Simon states that he failed in detecting any certain indications of albumen in the sweat collected (by means of linen washed with distilled water) from the breast of a person in the colliquative stage of tubercular phthisis.

is marked, the movements are languid, the respiration often embarrassed, and the mind depressed. The character of debility is strongly impressed upon them, and there is much correctness in the common feeling, that when dropsy appears, it announces the approach of decay, and of the breaking up of the system as it is called. One of their most common, but as we shall see, probably, not their immediate cause, at least in many instances, is, beyond doubt, a mechanical impediment to the free course of the blood. This was long ago proved by the well-known experiment of Lower. He tied the jugular vein of a dog, and found that all the tissues of the head and face were infiltrated after the lapse of some hours, not with extravasated blood, as he had rather expected, but with clear serum. The analogous instance of ascites resulting from cirrhosis of the liver has been already mentioned. But it requires no very long pathological experience to discover that there are cases not unfrequently occurring, in which, although there exist abundant causes of obstruction to the circulation, yet dropsy does not take place. Dr. Walshe, in his work on diseases of the lungs and heart, p. 478, has given a list of most serious diseased conditions of the heart which may exist without producing dropsy, and concludes justly, that something beyond all these is wanting to insure this occurrence. What, then, is this? Andral shows, in his *Hématologie*, that neither diminution of the globules, nor of the fibrin of the blood, is the immediate cause of dropsy, but, that this always accompanies a diminution of the albumen. It is, therefore, highly probable that it is this alteration of the crasis of the blood which determines the occurrence of dropsy in persons who are predisposed to it by organic disease of the heart or lungs, which causes congestion of the venous system. It is not only in dropsies of cardiac origin that diminution of the albumen seems to be the most important moment[1] in producing the effusion. This cause is evidently influential in renal dropsy, in which a constant drain of albuminous serum out of the blood is taking place. It is remarkable that this may have been going on for some, perhaps a considerable time, and yet no dropsy occur. The explanation of this is afforded by the circumstance ascertained by Simon and Christison, that the decrease of the solid constituents of the serum is not always the leading character in this disease. In three of Simon's analyses out of four of blood in Bright's disease, the quantity of albumen was decidedly increased—in one instance amounting to 109.4, considerably above the average of health. Cases of dropsy occasionally are met with, in which, as there appears no absolute organic disease, but only an hydræmic condition of the blood, one is obliged to conclude that the effusion is dependent on this. A female is now under our observation, in St. Mary's Hospital, who has considerable anasarca of the legs and feet, with puffed eyelids, without any discoverable albuminuria, or disease of the heart. Andral mentions that, during a famine, where the poorer classes had been obliged to seek a scanty nourishment in roots and herbs growing in the fields, many persons became dropsical. In this, and similar recorded

[1] We ask leave to introduce this word as a convenient term, signifying an influential condition.

instances, it is very probable that the proportion of albumen in the blood was diminished, as it is clear that the supply of it ordinarily derived from the food was so. When, from cardiac or renal causes, or both combined, together with altered crasis of the blood, the tendency to dropsical effusion is very strong, it is quite remarkable how universal the dropsy becomes; the peritoneum, both pleuræ, and the pericardium, may be found full of fluid, the areolar texture everywhere infiltrated, the air-cells of the lungs loaded with frothy serum, the tissue of the brain "wet," and the subarachnoid fluid considerably increased. In fact, it seems as if the vessels no longer presented any containing barrier, but permitted the escape of fluid in every part that it traversed. It is often observable in these cases after death how the naturally transparent serous membranes have lost this appearance; they look thickened, of a dull, white gray tint, as it were sodden in the fluid. There can be no doubt that this depends on a chronic thickening and increase of their fibrous layer.

We have next to examine the *composition* of dropsical effusions; this generally approaches more or less closely to that of the serum of the blood. The purer fluids are clear, tolerably limpid, and colorless; often, however, a marked yellow tint is observed, which may either arise from dissolved hæmatin, or from the presence of an increased quantity of the natural yellow pigment of the serum; or again, especially in the case of ascites, from dissolved bile pigment. If the latter is the case, it will be rendered evident by the reaction with nitric acid. "A large quantity of albumen (Vogel says) renders it (the effused fluid) viscid; a very large quantity, above twelve per cent., renders it thick, tenacious, and capable of being drawn out in threads, like albumen itself." A milk-white turbidity is occasionally observed, which depends on the admixture of fat (oil), or epithelium scales. Blood-globules may be often seen in great numbers in the fluid of ascites by the aid of the microscope: cholesterin tablets are common in that of hydrocele, and may be sufficiently numerous to constitute a crystalline deposit. The reaction of the fluid is alkaline; in rare instances an acid has been observed; in Vogel's opinion, probably from the presence of lactic. We subjoin, from the same author, the results of seven analyses, which show some remarkable variations in the amount of the several constituents.

	1	2	3	4	5	6	7
Water . . .	905.0	920.0	927	946	956	988.0	704
Albumen . .	78.0	71.5	48	33	29	0.9	290
Extractive matter	4.2		10	13	9		2
Fat	3.8		9		7	10.0	
Salts . . .	9.0	8.5	6	8	8		4
	Blood Serum.	Hydrocele.	Hydrocele.	Ascites.	Ascites.	Ascites.	Ascites.

The seventh analysis shows actually a larger amount of albumen than is present in the serum of the blood; this might be supposed to be an error if other similar instances had not been observed; it probably depends upon a quantity of the water of the original effusion having been removed by absorption, so that the fluid became more concentrated. Urea has frequently been found in dropsical fluids; its quantity is sometimes very small, sometimes amounts to 6 parts per 1000.

This seems to be the most proper place to introduce a short notice of certain *gaseous* effusions which there is good evidence to show take place occasionally in different parts of the body. To them the name of *Pneumatoses* has been given by Frank and others. Frank gives cases to prove that the subcutaneous cellular membrane, when slightly inflamed, may secrete air in abundance, and thus give rise to emphysema. The same has been observed after attacks of profuse hemorrhage during life,[1] and we are convinced we have witnessed something of the same kind in post-mortem examinations when there was no trace of putrefactive change. Dr. Graves, whose interesting article on the subject we need only refer to, mentions a case quite conclusive, as we think, of the secretion of gas to a considerable amount in the cavity of the pleura, and we have ourselves observed a somewhat similar occurrence. In the post-mortem examination of a female, who died with extensive bed-sores, after symptoms of fever, the right lung was found compressed against the V. column, and bound down by layers of false membrane, which also formed bands crossing the pleural cavity. This was *empty* of fluid, but must have been filled by air, as a very considerable space intervened between the lung and the wall of the thorax. No vomica was found in the lung, or any evidence of rupture of the pleura. The peritoneal cavity seems also to be the seat of gaseous accumulation in rare instances. The development of gas in great abundance from the mucous membrane of the stomach and intestines is a phenomenon of daily and troublesome experience; we speak now of an actual secretion from the mucous surface itself, and not of the result of decomposition of ingesta. The influence of certain states of the nervous system upon the development of gas is often evidenced in hysteric and emotional excitement, and also by the circumstance which we have observed, that it will take place, sometimes, very rapidly from the stomach after an action of the bowels. The accumulation of gas is sometimes so considerable as to produce alarming or very distressing symptoms; we have seen it even, in a minor degree, induce or aggravate attacks resembling angina pectoris.

A very remarkable case has been recorded by Sir F. Smith, of excessive development of gas from the stomach, also from the urinary bladder, and from the surface of the skin. Respecting the cause and mode of production of these pneumatoses we are totally ignorant, but the fact of their occurrence is most important in its relation both to practice and science.

INFLAMMATION.

We proceed, according to our plan, to the third variety of hyperæmia, that in which the movement of the blood in the part affected is partly increased, partly diminished. The truth of this definition, so far as it goes (though we believe it to be a very imperfect one), is shown—1st,

[1] A portion of the liver of a man, who died suddenly with aneurism of the left carotid, became so full of gas about two days after death, that it floated on the surface of water. Portions of the heart and kidney were not thus affected; they sank in water.

as to the increased motion by Mr. Laurence's well-known experiment of drawing blood at the same time from an inflamed and from a healthy arm of the same patient, when three times the quantity of blood flowed from the vein of the inflamed as from that of the healthy limb; and, 2d, as to the diminished motion, by looking at an inflamed part through a microscope when the arrest, or stasis, as it is called, of the blood current in the affected part is most evident.

The general appearance of an inflamed part is well described in the terms handed down from the age of Celsus, as being the seat of redness, heat, pain, and swelling. These are the visible symptoms of a pathological process, which, though continually before our eyes, and of the utmost importance in its results, and though it has been the subject of numberless speculations and careful labors, we are compelled to acknowledge we are still imperfectly acquainted with. The redness of an inflamed part is more or less vivid; it is deepest in the centre, and gradually shades off towards the circumference; in this respect it differs from an extravasation whose margin is more defined, as also in the circumstance that it can be in a greater degree removed by pressure, though by no means completely. The aspect of the redness may differ according to various circumstances; if the capillary networks of the part affected be a plane, or uniformly extended, the injection will appear as an uniform deep blush; if, on the other hand, they are moulded to the form of villi, or folds of mucous membrane, the surface will have the appearance of a pile of red velvet: in fibrous structures a streaky appearance is observed, and generally the form of the redness will depend upon the arrangement of the capillaries of the part. The increased depth of color is owing chiefly to distension of existing vessels, not in anywise to the formation of new ones, a process which does not take place till a much later period: the only other cause which at present exists is the staining of the surrounding tissues with exuded hæmatin, which may occur soon after stasis has been established. It is necessary to distinguish carefully between genuine inflammatory redness, and that which often simulates it closely in the dead body, viz: hypostatic, or depending solely on mechanical causes, or on the mode in which death has taken place. It is always desirable to take other circumstances into consideration at the same time, but we may generally say that we should suspect the inflammatory nature of a redness which existed solely in depending parts, or in those the large veins of which were much gorged, or which coincided with a fluid condition of the blood, and which was not attended with any thickening of the part. It may also be observed, that after an internal part has been exposed to the air a short time, it assumes a much more marked and brighter redness, which depends solely on the action of the oxygen in the air.

The natural *temperature* of an inflamed part seems to be considerably increased, and this as well to the sensations of the observer as of the sufferer (hence the name "inflammatio," a burning). An increase of heat is, however, not so decidedly shown by the thermometer; in some of Hunter's experiments the difference was not more than 1°, and it seems doubtful whether the heat of the inflamed part is ever greater than that of other parts of the body. The highest temperature we find

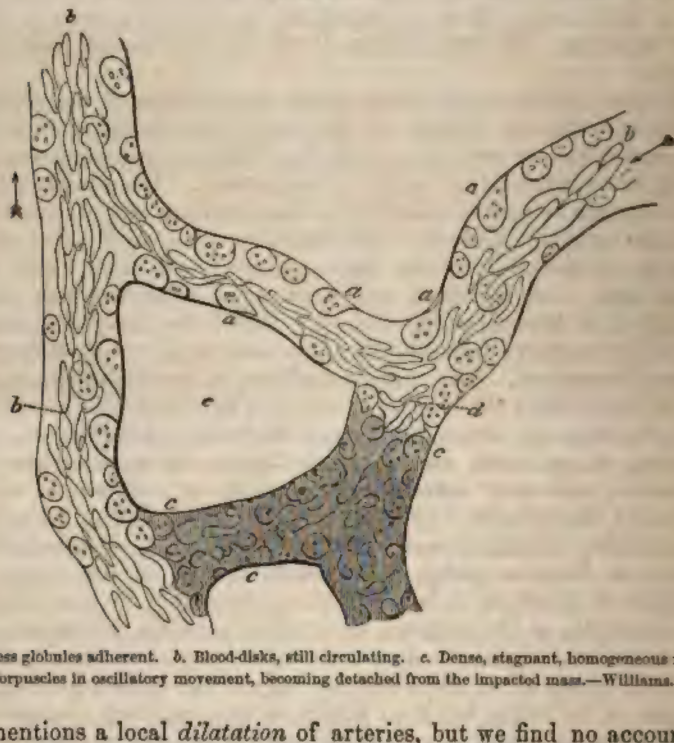
mentioned is 110⅘; this was in tetanus, which is not an inflammatory disease. Rokitansky says, that the increased temperature in inflammation is partly occasioned by the formative processes which take place in the stagnating blood, but that a very important part in the phenomena is also played by the excitation of the sensitive nerves. It is this nervous excitement, doubtless, which gives rise to the idea of the great increase of temperature. The *pain* of inflammation varies much in degree and in kind, according to its seat, and intensity, and exciting cause. That of inflamed serous membranes is often of a peculiar, sharp, darting kind; that of mucous membranes more dull and gravative, as it is termed; that of dense, unyielding, fibrous or bony textures, amounts sometimes to extreme agony. The inflammation heightens the sensibility of the nerves, which are at the same time compressed by the swollen textures and distended vessels.

Swelling depends manifestly in great measure upon the distension of the vessels with blood; the bulk of the part is increased just as that of a kidney or liver is when it is injected artificially—its fibres are put on the stretch, its vessels strained, and its capsule, if it have one, fully distended. The effusion of plasma is considered by Rokitansky as the principal cause of the swelling; this may be the case at a later period, and in some cases, but is not, we think, so powerful a cause as the vascular injection. In loose textures it will be more considerable than in denser.

We now proceed to the more minute examination of the phenomena of the inflammatory process as they have been disclosed to us by the microscope, and we here resume the line of inquiry which we commenced during our consideration of active hyperæmia. We saw in this, that, with a certain amount of stimulation, the arteries enlarged, and admitted a greater quantity of blood, which flowed on more rapidly, and traversed with an accelerated current the capillaries and veins which became dilated also. An increased and more rapid blood-flow were then the characters of determination of blood. But if the stimulus is increased, or if it be excessive from the first, phenomena of a very different kind present themselves. The current slackens, it moves slower and slower, and at last ceases; the capillaries are seen distended with a red uniform mass, the veins are also enlarged, and filled with red corpuscles, crowded together, which retain more of their distinct form than those in the capillaries, and move either slowly onwards, or oscillate, or are quite stagnant; the arteries, which are also distended, exhibit for some time a progressive movement onward of their contents, which at first is steady, afterwards becomes jerky, or intermittent, and at last ceases. The condition of *stasis*, as it is called (ἵστημι, to stand), is now established, and therewith, as the sequence of active hyperæmia, inflammation. In the immediate neighborhood of the seat of stagnation the circulation is still seen going on rapidly, and not only in the parts adjoining on the margins of the stasis, but even within its area capillary streams may be seen here and there rapidly coursing beneath a plexus of channels, which are filled by an uniformly red quiescent mass. Manifestly, determination of blood prevails actively all round the focus of arrest of movement. The stagnant blood in most of the capillaries presents an uniform red

mass, in which the separate corpuscles are undistinguishable; here and there gaps may be seen, as if a fissure had taken place, and separated the adjacent portions a little away from each other. Amid the mass of blood-globules, appearing as it were fixed together, may be seen occasionally one or two white corpuscles; according to our observation, they are certainly not to be seen in the great majority of capillary vessels. After the stasis has existed some time, they may be seen in great numbers coating the walls of the veins, and rolled along by the current passing through them; and occasionally they constitute, together with transparent plasma, the entire contents of a portion of vessel of some length, not remaining absolutely stagnant, but oscillating to and fro, or moving sometimes slowly onwards. From a vessel thus filled we have observed them escaping into a communicating vein, three or four at a time, and carried away into the general circulation. The diameter of the distended capillaries, which are the seat of stasis, has appeared to us, for the most part, tolerably uniform, but in one instance we observed numerous constrictions at various points. These were remarkably abrupt, and extended across one-third, or one-half, the channel. Mr. Wharton

Fig. 11.



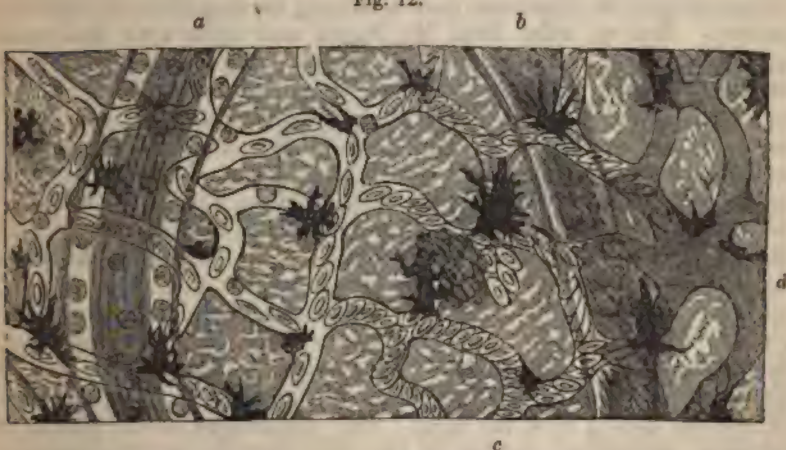
a. Colorless globules adherent. *b.* Blood-disks, still circulating. *c.* Dense, stagnant, homogeneous mass. *d.* Corpuscles in oscillatory movement, becoming detached from the impacted mass.—Williams.

Jones mentions a local *dilatation* of arteries, but we find no account of these local contractions of the capillaries. The red globules, for the most part, appear to be packed together without any regular arrangement, but occasionally they may be seen lying together in rouleaux,

like the corpuscles of human blood, with their long diameter transverse to the axis of the vessel. The white corpuscles are not unfrequently seen of a pyriform shape, dragging slowly along, or actually sticking to the sides of the vessels; that they do possess some degree of adhesiveness is manifest, but it does not seem to be so considerable or general as Dr. Williams supposes. When the inflammation is subsiding, and the stagnant blood beginning again to resume its course, all that can be observed is, that the agglomerated mass of red corpuscles in a vessel loosens and breaks up, so that the individual corpuscles are again visible, while the impulse of the heart makes itself more and more felt, and at last sweeps away the accumulation altogether, having first detached small portions successively. Fibrinous coagula also form occasionally, as Mr. Wharton Jones describes, and are similarly disintegrated, and carried away by the returning current during resolution of the inflammation. While stagnation continues, a small quantity of hæmatin dissolved in the serum exudes, and imparts to the tissues bordering the vessels some degree of yellow staining.

We intend the foregoing account to serve as a description of what may

Fig. 12.



An exact copy of a portion of the web in the foot of a young frog, after a drop of strong alcohol had been placed upon it. The view exhibits a deep-seated artery and vein, somewhat out of focus; the intermediate or capillary plexus running over them, and pigment-cells of various sizes scattered over the whole. On the left of the figure, the circulation is still active and natural. About the middle it is more slow, the column of blood is oscillating, and the corpuscles crowded together. On the right, congestion, followed by exudation, has taken place, constituting inflammatory action in the part.

a. A deep-seated vein, partially out of focus. The current of blood is of a deeper color, and not so rapid as that in the artery. It is running in the opposite direction. The lymph-space on each side, filled with slightly yellowish blood-plasma, is very apparent, containing a number of colorless corpuscles, clinging to or slowly moving along the sides of the vessel.

b. A deep-seated artery, out of focus, the rapid current of blood allowing nothing to be perceived but a reddish-yellow broad streak, with lighter spaces at the sides.

Opposite *c*, laceration of a capillary vessel has produced an extravasation of blood, which resembles a brownish-red spot.

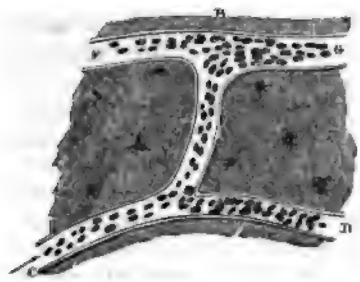
At *d*, congestion has occurred, and the blood-corpuscles are apparently merged into one semitransparent, reddish mass, entirely filling the vessels. The spaces of the web, between the capillaries, are rendered thicker and less transparent, partly by the action of the alcohol, partly by the exudation. This latter entirely fills up the spaces, or only coats the vessel.—Bennett.

be actually observed of the process of inflammation, as it occurs in the frog's web. Most of the statements we have verified by our own examination, and we believe they are in accordance with those of the best observers. The question now presents itself, what occasions the stasis? and this is, in fact, the great and unsolved problem of inflammation. The earlier speculations as to the vessels being in a state of spasm, or, on the other hand, of atony, need not occupy our attention; nor can we see that any definite ground is gained by the hypothesis of Hunter, that the vessels are in a state of active dilatation, except that he seems thereby to recognize the true nature of active hyperæmia. Three opinions, of later date, may be said to be still *sub judice*. Two of them we have already referred to, when speaking of active hyperæmia. The third is that maintained by Dr. Williams. He considers "that an essential part of inflammation is the production of numerous white globules in the inflamed vessels; and that the obstruction of these vessels is mainly due to the adhesive properties of these globules." With regard to this doctrine, a reference to the description we have given will show that we agree with Professor Paget and Mr. Wharton Jones, in rejecting it. Nothing can be more evident, we think, than that the white corpuscles play no important part in causing the stagnation of the blood-current. But we differ from Mr. Paget, and agree with Rokitansky and Williams, as to the increased production of white corpuscles in the inflamed vessels. The numbers which present themselves in some of the small veins, cannot, we think, be brought together merely by accumulation, especially when we consider that most of the communicating capillaries are plugged up, and that they cannot therefore be transmitted from them. The neuro-pathological theory of Henle is the one which Rokitansky prefers; but he acknowledges that it gives no satisfactory explanation of stasis of the blood. Henle himself thinks he can account for it according to his view as follows:[1] "As a physical consequence of dilatation of the vessels, there takes place a retarded flow of blood. This, together with the relaxation and dilatation of the vessels, favors the exudation of serum; the consequence of which is, that the plasma of the blood in the part becomes inspissated by a preponderance of protein matter over the salts. This inspissation of the plasma determines endosmotic changes in the red corpuscle, in consequence of which they are disposed to aggregate." Rokitansky, stating that mere paralysis and dilatation of the vessels cannot give rise to stasis, gives the following as his own opinion of its mode of production: "It proceeds (*a*) from the sticking together of the blood-corpuscles, the heaping up and wedging together of them in the capillaries, while the plasma in part flows off towards the veins; (*b*) from the inspissation of the plasma occasioned by the exudation of serum through the dilated and attenuated walls of the vessels, and its saturation with fibrin and albumen; (*c*) from the heaping up of the colorless corpuscles—*i. e.* nuclear and cell-formations, together with blood-globules; from their sticking together, and from the delicate, hyaline, fibrinous coagula, which develop themselves among them. This is certainly the most important

[1] Wharton Jones's Report on Inflammation, April, 1844.

moment in the inflammatory process, since, on the one hand, it very specially throws light upon the phenomenon of stasis, and, on the other hand, comprehends also the plastic processes which take place in the heaped up and stagnant blood. It separates in this way the process of inflammation from a merely simple one of exudation. The elementary formations above mentioned are not merely swept together towards the demesne of the stasis, but they originate as new (productions) in the stagnant blood, for this generally presents remarkable alterations. . . . ." We believe the latter paragraph to contain views of very great importance, especially with regard to the effects of local inflammation upon the system. Nor do we doubt that the changes wrought by exudation, draining away of Liq. Sanguinis, multiplication of white corpuscles, and coagulation of the fibrin, may all exercise considerable influence in prolonging and confirming the stasis, and determining its results. But we do not think they are the primary and causative phenomena. It seems to us a very important fact, which Mr. Wharton Jones states, and which we have observed ourselves, that "stagnation commences in the capillaries, and extends from them to the veins on the one hand,

Fig. 18.



Production of stasis from Mr. Wharton Jones's Essay, Guy's Hospital Reports, vol. vi. p. 35.

and the arteries on the other." We have seen the blood stagnant in the capillaries, while it was moving on steadily through an adjacent artery and vein. This points to the capillaries as the part where the arrest commences. Again, it is a very important circumstance, that, if a strong stimulus be applied, the stasis takes place almost immediately: it seems as if the blood were suddenly coagulated in the capillaries. When the arrest comes to pass more slowly, so as to admit of being watched, it is described by Mr. Wharton Jones as taking place in the following way: Red corpuscles, more collapsed and darker-looking than natural, first adhere to the walls of the vessels, and then other red corpuscles adhere to them. "The first adhesion of red corpuscles to the wall of a vessel usually takes place at a bifurcation, and in this manner (see Fig. 6): The stream C, striking with force on the wall of the vessel, at the bifurcation A, some of the red corpuscles adhere to the wall of the vessel. Other red corpuscles adhering to them, an agglomerated mass results, which is sometimes seen to be moved along the vessel a short way by the force from behind; but more red corpuscles adhering

to the mass, the vessel is at last wholly blocked up by it at D. The stream C, being no longer permitted to pass in part by D, passes off by E; but in leaving E, the stream strikes on the wall of the vessel at the bifurcation B, where red corpuscles adhere, and form a nucleus for an agglomeration, which blocks up both the vessels F and G—E and C also becoming blocked up, and so on the process goes. Stagnation is seen first to take place in those capillaries which are least in the direct course from the artery to the vein. In those capillaries which lead most directly from the artery to the vein, and in which, consequently, *vis à tergo* operates most advantageously, the blood is latest in stagnating." The main point, that stasis is produced by the red corpuscles adhering to the walls of the vessels and to each other, is confirmed by others, and by our own observation. On one occasion, we distinctly noticed a single red corpuscle adhering, by one end, to the wall of a vessel, in which circulation was returning, while several other red corpuscles, in motion one after another, swept slowly past, brushing against it, as it waved in the current. That in the healthy state the red corpuscles pass on so smoothly and uninterruptedly within their even relatively narrow channels, that they shun, as is well known, the walls, and allow a thin layer of the fluid in which they float to intervene, while the colorless corpuscles show no such tendency, but affect a preference for the so-called still layer, and move slowly onwards in it; that there is decidedly a tendency of the red globules to aggregate together in blood drawn from the body, while this seems to be held in abeyance, while the blood is within the vessels; that the smallest capillaries, according to Wharton Jones, are traversed chiefly by plasma, and by a few colorless corpuscles, one after the other, with only a single red corpuscle now and then; that the red and colorless corpuscles show no tendency to stick together, appear to us very significant facts, which require some such hypothesis as that offered in Wharton Jones's report, April, 1844, viz: "That there exists some sort of attraction between the colorless corpuscles and the walls of the vessels, but an absence of attraction, if not a repulsion, between the red corpuscles and their walls, as also between the red and colorless corpuscles." Mr. Wharton Jones formerly conceived that it was the nervous influence which prevented the red corpuscles from aggregating together within the vessels, as they do out of the body, and that stasis depended on the suspension of this influence. It was an ingenious and probable opinion, but is disproved by the experiments which he himself has performed, and which are related in the Astley Cooper Prize Essay. In these he shows, that, after the nerve accompanying an artery had been divided, the flow of blood, though interrupted for a short time, soon returned, and became quite free; and also that, after section of the ischiatic nerve, the arteries of the web of the same side are found somewhat more dilated, and the flow of blood in them freer and more rapid than in the web of the uninjured side, while stagnation was induced more readily in the web of the limb whose nerves were entire, than in that of the limb whose nerves had been divided. He now adopts the opinion of Henle, though somewhat modified, that stagnation depends on inspissation of the plasma, on its containing an increased quantity of fibrin and albumen,

and rests much on the fact that such a condition of the Liq. Sanguinis, whether natural or imitated artificially, is found to increase the tendency of the red corpuscles to aggregate together. Our limits forbid discussion; and we therefore simply pass on to state our own opinion, so far as we may venture to offer one on this *quæstio vexata.* We saw reason to believe that the tissues, in yirtue of their nutrition power, exercised an influence on the movement of the blood; that in active hyperæmia their attractive force was increased; and we would now add, that it is through the failure of this nutrition power that we believe stagnation takes place. The exact nature of the influence exercised by the tissues over the blood, which traverses the capillary channels, is unknown. All that we can discern is, that it is such as promotes its free passage through them; and therefore, when it is in abeyance or greatly altered, it is to be expected that the circulation will be interrupted also. More than this we cannot gather from the observed phenomena; and we would only offer the remark, in conclusion, that in coincidence with the establishment of complete stasis, cessation of the natural function of the part occurs, and other processes commence—the exudative, in which the plasma, that in a healthy state would have ministered to and maintained healthy action, is consumed in wasteful or even destructive changes. Whether the stasis depend solely on a persistence and exaggeration of the attraction of the tissues for the blood which exists in active hyperæmia, or upon this and an abolition of the natural non-aggregative tendency, or even repulsive tendency of the red corpuscles for each other and for the walls of the vessels, must remain uncertain; but the coincidence above noticed must be allowed to give considerable support to the main point, on which we would insist, viz: that the nutrition power of the tissues is chiefly concerned in the production of the flow of active hyperæmia, and the stasis of inflammation.

We must take some notice of the different varieties of inflammation, which depend, partly on differences inherent in the subject, partly on different exciting causes. What is called *Sthenic* inflammation is that which occurs in a healthy person, either spontaneously, or from exposure to cold, or the application of some irritant which does not contaminate and depress the general system. The symptoms, if the inflammation is extensive, run high, the febrile excitement is considerable, and free depletion is required and borne well. The exudations contain much plastic matter, and pus, if formed, is of the kind termed *laudable.* *Asthenic* inflammation occurs in persons originally weakly, or rendered so by the action of the exciting cause, as in the inflammations of influenza; the pulse, though frequent, has no strength, the fever is of a lower type, and the effused matters manifest little plasticity. The terms *acute*, *sub-acute*, and *chronic*, have reference mainly to the periods of duration of the inflammation, or to the rapidity or slowless of its course. Acute inflammation is often, but not necessarily, sthenic; chronic and sub-acute are often not asthenic. Many changes are commonly said to proceed from chronic inflammation which probably belong more to the class of degenerations: it is, however, difficult to draw any marked line between the two. The chief value of the terms lies in their

affording a kind of scale whereby to apportion the activity of treatment; to the chronic affection we must oppose a remedy of slow and gradual action: the acute must be met more "heroically." It is important to be aware that an inflammation may be sub-acute or chronic from the outset; an acute inflammation cannot well be overlooked—a chronic, if unpreceded by acute symptoms, may easily be: such inflammations are sometimes said to be *latent*. Congestive inflammation differs not much from asthenic; its effusions are of the same kind, but it partakes in a considerable degree of the nature of passive hyperæmia—indeed, is such originally, and has, subsequently, inflammation, acute or sub-acute, grafted upon it. This must be remembered in treating it. The chief character of erythematic or erysipelatous inflammation is its tendency to spread and travel over an extensive surface: this seems to depend, at least in part, on the peculiar character of the effusion, which consists, for the most part, of serum, or sero-purulent matter, and not of fibrin, which in phlegmonous inflammations establishes a barrier between them and the surrounding textures. The general symptoms are in most cases those of adynamic or typhoid fever; depletion is injurious, and stimulants required at an early period. There is good evidence to show that a peculiar poison, capable of being communicated by infection, is the cause of these inflammations, and that this acts upon and modifies the system, even before the phlogistic process has made its appearance. They are, therefore, with respect to their exciting cause, to be ranked together with other inflammations, such as the rheumatic, gouty, syphilitic, &c., which each manifest certain peculiarities, but depend, essentially, upon the presence of some *materies morbi* in the blood. To the same class belong most of the inflammations which constitute skin diseases, which exhibit very remarkable instances of the effect of different states of the blood in determining the kind of inflammation that shall occur. To this we shall refer again, under the head of "Crasis." These inflammations also exhibit very clearly the affinity of certain parts of the tissues for certain morbid matters, which are their exciting causes; thus, lepra has its seat of election about the prominence of the knees and elbows, eczema prefers the side of flexion of the limbs and the bends of joints, lichen affects the outer sides. The same is exemplified in the action of many medicines and poisons; arsenic, in small doses, produces conjunctivitis—in larger, inflammation of the stomach and intestines, corrosive sublimate inflames the larger intestines, mercury, the gums, and so on: in all these cases the affinity or attraction of the elements of the tissue for the substance is clearly evinced. *Diphtheritic* inflammation is characterized by the early exudation upon mucous surfaces of a film or membrane of fibrinous matter of dirty white or grayish appearance; this may extend over a considerable tract, commencing often in the fauces, and thence spreading to the mouth, the larynx, the air-passages, the œsophagus, and more or less of the alimentary canal. The subjacent mucous membrane is but little swollen, of a deep dull red, and inclined to bleed on the removal of the exudation. The attendant fever is of a low kind, and much of the danger depends upon the insidious, almost latent manner, in which the exudation takes place, so that suffocation may be actually threat-

ened before alarm is taken. This kind of inflammation occurs most often in epidemics, and is more frequent on the Continent than in England. It has been observed in France, that, during its prevalence, wounds and ulcerations assumed an unhealthy character, and were indisposed to heal. Its cause is, evidently, a peculiar asthenic state of the system generally, involving a peculiar crasis of the blood, induced by atmospheric influences. Instances of a somewhat similar kind, but in which the asthenic character is much less marked, are occasionally met with among ourselves; among these we should rank the so-called bronchial polypi, the pieces of membrane which are passed after the irritation of calomel, and in some other cases of intestinal disorder, and, probably some of the membranous exudations of dysmenorrhœa. The aphthæ of children and adults belong more to the true diphtheritic exudations; they contain, often, a large proportion of a confervoid growth, which has been regarded, indeed, as constituting their essential cause. This, however, in Rokitansky's opinion, is not the case. To this subject we shall refer again, under the head of "Parasites." *Hemorrhagic* inflammation is another variety; it seems chiefly to occur in individuals predisposed to hemorrhage, or in places where scurvy is prevalent. Dr. Williams has found it associated with cirrhosis of the liver, and granular degeneration of the kidney. His opinion is, no doubt, correct, that it is more dependent on an altered condition of the coloring matter, than on a deficiency of the fibrin. Its character is decidedly asthenic. *Scrofulous* inflammation is not so much a distinct variety as some others. It is commonly excited by the irritation of existing tuberculous deposit, which is occasionally mingled with its exudative products. These are distinguished in general by their aplastic character; they are deficient in coagulating fibrin, and are often thin and serous. The vital power of the system, both blood and tissues, is essentially defective; and is the cause, at once, of the deposit which excites the inflammation, and of the low character of the process itself.

We have already alluded to the *causes* of inflammation in the foregoing remarks, and shall now do little more than briefly enumerate them. Predisposing causes are almost always debilitating influences; a strong part is less liable to inflame than a weak one—a previous attack of inflammation especially renders a part more prone to undergo a second. Certain unhealthy conditions of the blood (of which that induced by foul air is one) predispose the system to inflammation from trifling causes, which would pass inoperative in a sound state. Exciting causes are either such as act on the part directly which they inflame, or indirectly through the medium of another. The first may be *mechanical* irritants, such as a splinter in the flesh; or *chemical*, as a strong acid, or acrid salt; or *vital*, such as mustard, &c., whose operation only affects living structures. It is remarkable that the urinary and biliary secretions which excite only healthy action in the mucous surface over which they naturally flow, and which, or at least some of their constituents, produce no particular injurious effects when absorbed into the blood, act as the most violent and fatal irritants upon serous membranes, and the areolar tissue, when infused into them. This shows

clearly the important part played by the tissues themselves in the process of inflammation: that which is a healthy stimulant to one texture is the cause of destructive inflammation to another. The production of local inflammations from the presence of some substance in the general mass of blood, for which certain parts seem to have a special affinity, has already been noticed, but we may add, that it is in these cases that we observe the interesting phenomena of symmetrical disease;[1] the corresponding parts of the two lateral halves of the body being affected almost to the exclusion of others. Here, again, we have evidence of the predominant influence of the tissues, the parts which are exactly alike are affected alike, and the *materies morbi* passes by others. How unable is the neuro-pathological theory to explain such instances of inflammation!

The second class of causes are those which act indirectly on the part which suffers. The most common of these is cold, which appears to act by repelling the blood from the surface, and causing it to accumulate in some internal part. This will be different, according to the previous predisposition; thus one person, as the result of a severe chill, will have bronchitis, another diarrhœa, a third peritonitis, a fourth renal congestion, and so on. Malaria, the repelling of eruptions, the arrest of habitual discharges, the sudden healing of ulcers, are also recognized as causes of internal inflammations, which they probably produce in the same way as cold, but the active congestion of the incipient process is more apt to issue in hemorrhage. It is matter of much uncertainty as to how many inflammations originate; they come on, as it seems, spontaneously, without the individual being aware of any exciting cause. Both as respects these, and those which are produced by cold, &c., it seems to us necessary to recognize some special condition of the tissue, which in the one case converts the congestion into an inflammation, and in the other, is the sole and efficient cause. In ague, for instance, during the cold stage of each paroxysm, considerable congestion of the internal viscera takes place, but inflammation of these is comparatively rare. In healthy states of the system, the surface may remain severely chilled for several hours, during which the blood must accumulate in the internal organs, but this does not occasion inflammation. These instances show that mere repellent influences producing congestion are not adequate to produce inflammation solely by themselves. Even in the case of a common catarrh there is much reason to believe that the inflammation of the mucous surface depends much more on some preexisting dyspepsia, or unhealthy condition of the blood, than on exposure to cold or wet; and, in fact, many a cold occurs without any such exposure. In such cases, the predisposing cause becomes the most important, or may even be the exciting also. We have not yet noticed the nervous influence in its relation to the causes of inflammation. This is considered the prime mover in the process by those who adopt the neuropathological theory: in our view it holds a very secondary place. That disturbance of the nervous force *may* prove a cause of inflammation,

[1] For a most interesting exposition of the subject of symmetrical diseases, we refer to Mr. Paget's Lectures for 1847.

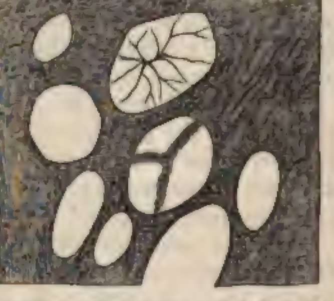
cannot be doubted: Lallemand (quoted by Dr. Williams) "relates a case in which a ligature, involving the right brachial plexus, was followed by inflammation and suppuration of the opposite hemisphere of the brain. Mr. Paget mentions a case in which a portion of a calculus impacted in the urethra excited inflammation, with deposits of lymph and pus in the testicle. Instances of a similar kind, or of active hyperæmia similarly occasioned, are not very rare, and they certainly prove the capability of the nervous influence to set on foot the inflammatory process. But we have already given abundance of evidence that it is not through this channel that the causes of inflammation usually operate, and for a summary of the arguments we would refer to Dr. Williams's work, p. 249. Coinciding, as we completely do, with Mr. Paget, in his enumeration of four conditions as necessary to healthy nutrition, and believing that the derangement of one of these primarily, with secondary derangement of the others, occurs in every case of inflammation, we might divide the various causes which we have noticed into such as affect—(1) the contractility of the vessels; (2) the healthy crasis of the blood; (3) the nervous influence; (4) the life and nutritive actions of the part.

In strict language, there is only one termination to the inflammatory process, viz: that which is commonly called resolution, in which the diseased action ceases to advance, and then recedes by the same steps as those by which it arrived at the condition of stasis. The microscopically visible phenomena have been before described, and they correspond to the subsidence of the general symptom, to the paling of the redness, the lowering of the temperature, the lessening of the swelling and pain. The recovery of the part may be complete; but more often some, it may be slight, indications remain, for a time, of the by-past malady, and of some deficiency of the vital powers. These consist, in some degree, of congestion of the vessels, especially the veins, from an enfeebled state of their contractility, in a less perfect fulfilment of the function of the part, and in a proneness to relapse on the application of slight exciting causes. Inflammations which arise in consequence of a mal-crasis of the blood, rarely undergo resolution, or if they do, it is only to reappear in another part, and perhaps a more important. This constitutes metastasis. A good example of this transfer of inflammation from one part to another is afforded by some cases of rheumatism, and occasionally by the disease called mumps. As long as the *materies morbi* continues to circulate in the blood, it will tend to excite inflammation in one part or other; the best thing that can happen is, that it should locate itself in a part where it can produce no serious effects from interference with important functions, and there remain until the dyscrasia is at an end. One important remark of Rokitansky's must not be omitted, viz: that even resolution does not, especially if the inflammation has been extensive, leave the system in as favorable a condition as before the attack, since a large quantity of liquor sanguinis, which, during the stasis, has undergone certain changes, is set free, to mingle with the general mass of the blood. This must produce a contaminating effect until it be eliminated. Hence the benefit of a free action of the skin, of a free flow of urine, purging, &c.

But in order that resolution may take place, it is absolutely essential that no considerable amount of effusion should have occurred; if this is the case, the affected tissue remains clogged and otherwise injured by the presence of solidified matter in its interstices. This matter and the tissue may also undergo further changes. We thus come to the consideration of, *firstly*, inflammatory exudations; and, *secondly*, of the changes that take place in the tissues affected by them. The fluid effused in inflammation is commonly the liquor sanguinis, more or less modified, especially containing a less proportion of albumen and fibrin. An analysis, by Simon, of fluid obtained by paracentesis thoracis, shows, in 1000 parts, 934.72 of water, 1.02 of fibrin, 1.05 of fat, 48.86 of albumen and albuminate of soda, 11.99 of extractive matter, and 9.5 of fixed salts. The quantity of fibrin varies considerably. Some effusions consist chiefly of it, others contain very little; generally, it may be said, its quantity is in proportion to the vigor of the system, and the acuteness and sthenic character of the inflammation. The fibrin may coagulate quickly, or remain in its fluid state, for a length of time, in the part where it is effused. Blood-corpuscles, or dissolved hæmatin, may be mingled with the exuded matter in various proportions. Though we often speak of serous effusions as the result of inflammations, yet the opinion expressed by Mr. Paget is probably correct, that "an effusion of serum alone is a rare effect of inflammation, and that generally it is characteristic of only the lowest degrees of the disease." He mentions as instances inflammatory œdema of the mucous folds above the glottis, chemosis of the conjunctiva, and some forms of hydrocephalus. The fluid obtained from blisters contains either distinct fibrinous coagula, or only a small proportion of fibrin, together with multitudes of puriform corpuscles.

No doubt, in inflammations of different characters, there must exist great differences in the composition of the serous fluid effused, and in its particular qualities and tendencies. There must be great variations

Fig. 14.



Fibrinous exudation on pleura in process of absorption; areolæ form in it, and reduce it to filamentous bands.

in the proportion of oil, of extractive and saline matters in the effusions; but respecting these we have scarce any information. The chemical examination of inflammatory products is very difficult, partly in con-

sequence of the impossibility of procuring more than very small quantities, partly because they can so seldom be obtained pure; and almost the whole of our knowledge, therefore, has reference to the differences which are perceptible to the eye and to the microscope in the solidified and shaped constituents of the exudation. These, however, afford very valuable indications for forming a judgment of the nature and tendency of the process from which they spring. We have already given Rokitanky's account of the varieties of fibrin, as seen in *intra-vascular coagula*, and stated the important circumstance that they correspond closely with those observed in exudations; but it still seems desirable to present an abbreviated sketch of the latter from the same authority, in order to furnish a kind of scale of varieties, to which observers may refer different specimens they meet with, and to elucidate, as much as possible, a process which meets us at every turn.

Rokitansky describes two varieties of fibrinous exudation, the *simple*, or plastic, and the *croupous*. The first appears as a flaky-fibrous transparent blastema, of remarkably sticky quality, tearing, as a fibrous felted mass, with numerous nuclei and nucleated cells scattered over it. It corresponds to the second variety of intra-vascular fibrin. Such an exudation constitutes the material by means of which wounds are united that heal by the first intention, and is often found forming the false membranes upon serous surfaces, or the induration-matter of parenchymata. It may undergo metamorphosis; (1) by being more or less entirely absorbed, in which Rokitansky considers the serous part of the exudation to act as a corrosive or dissolving menstruum; (2) by becoming obsolete, *i. e.* drying up into a horn-like mass, which may afterwards ossify; (3) by undergoing a change (of development) into fibroid tissue. New vessels may from in it more or less abundantly, or it may obtain a smooth, polished surface, like that of a serous membrane, as is occasionally seen in the arachnoid.

Fig. 15.

Copied from Gulliver's Trans. of Gerber, may be taken as an explanatory diagram of commencing organization in effused fibrin.

The *croupous* exudation is, in general, characterized by a high degree of coagulability, a yellow or greenish-yellow color, opacity, deficient capacity for becoming organized, speedy breaking up, and diffluence, very often by a corroding property, which brings the tissues, as it were, into a state of fusion.

The croupous exudation-process and its product are further remarkable by (1) the usually excessive, exhausting quantity of the exudation, and its extension over large surfaces of tissue; (2) by the acute occurrence of the exudation, since a pre-existing crasis is the fundamental cause of the stasis; (3) by the often very slight degree of injection of the diseased tissue, which may depend either on the blood-corpuscles being concealed by the opaque hyperinotic plasma, or on the large quantity of exudation, inducing quickly emptiness and collapse of the vessels; (4) by a less sticky quality; (5) by a considerable amount of fat. The principal alteration which this exudation undergoes is the above-mentioned diffu-

ence, by which it breaks up into a fluid, more or less analogous to pus. This change especially affects the coagulated solid part. When thus fluidified it may be absorbed, or may leave behind a partial residuum, of a cheesy, fatty, pappy fluid, containing granular, cretaceous, and oily molecules, granule-cells, and crystals of cholesterin. Variety (α) of the croupous exudation corresponds to variety (3) of the intra-vascular fibrin, like it consisting of varying proportions of felted fibres, punctiform and oily matter, and various granular nuclei and cells. Variety (β) corresponds to (4) of intra-vascular fibrin. It consists of a shapeless basis-substance, containing a preponderance of punctiform matter, together with nuclei and cell-formations, approaching more and more the character of those of pus. It is non-adherent to the surface, and quickly becomes diffluent. To these two varieties belong the various exudations of croup, those in many cases of meningitis, of pericarditis, and phlebitis. Most cases of pneumonia give rise to exudations of this kind, especially those in which a very abundant yellow, quickly-diffluent material is deposited, which causes great increase in the size of the lungs.[1] The corrosive quality of the diffluent exudations manifests a decided tendency to the early production of abscess and ulceration; it is probably in the case of such exudations as these that the comparatively rare occurrence of pulmonary abscess takes place. Variety (γ) of the croupous exudation, Rokitansky distinguishes as the aphthous, or, we may say, the diphtheritic. It forms a yellow or greenish-yellow mass, dirty-gray, and opaque, coagulating on surfaces into tenacious membranes, and thereupon breaking up, and occasioning corrosive fusion of the tissues. The affected tissue may be simply corroded, or fused into an ill-looking, stinking, sanious pulp, or into a tenacious, tinder-like, dirty scab. Instances of this exudation are seen especially on mucous surfaces, as in muguet, diphtheritis, some forms of dysentery, of puerperal metritis, in the ulcerations of typhus, and those of hospital gangrene. These exudations are, for the most part, dependent upon a special crasis of the blood, which we shall afterwards notice. Rokitansky makes a third variety of fibrinous exudations, viz: the *tuberculous*, but we shall not notice it in this place, as we doubt the propriety of classing it in any way together with the foregoing. There is, however, one form which fibrinous exudation not unfrequently assumes, which resembles tubercle a good deal, and might be confounded with it. In this it constitutes small, firm nodules, which consist of a fibroid tissue, and do not undergo any of those changes which tubercle commonly does. They lie sometimes in great numbers in the sub-pleural, areolar tissue, and are often surrounded with black pigment. Rokitansky also describes *albuminous* exudations, as distinct from the fibrinous, but the separation scarcely seems to us to be warranted. They appear identical with the lower varieties of the croupous form, but modified, in some degree, by occurring in depressed conditions of the system; as where the blood is abnormally venous, from disease of the heart, or impoverished by effusions that have robbed it of much of its fibrin, or insufficiently renewed, in consequence of marasmus,

[1] Dr. Hodgkin believes, and we think he is right, that this condition of gray or purulent infiltration is not preceded by a stage of red hepatization.

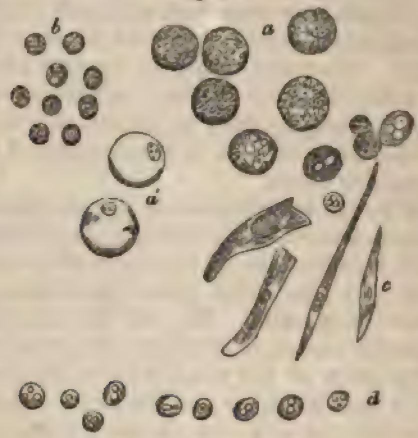
old age, &c. The stasis, which gives rise to these exudations, is said to have very often an asthenic, hypostatic character, and protracted course. We have above noticed the rather rare occurrence of serous exudations which contain no fibrin. Rokitansky denominates these *albumino-serous*, or, if they contain fibrin, *fibrino-serous*, reserving the term "serous" especially for those which contain little or no animal matter. Of these, he says that they are thinly fluid, watery, clear, colorless, or pale yellowish, or even yellowish-red, and of saline taste.[1] They may result from inflammation, but afford no proof by their presence of its having existed. They are, of course, capable of no organization, but seem to exert a deteriorating influence on the tissues where they are effused, "loosening and puffing them up, paralyzing their contractility, and manifesting, after long contact, a surprising enfeebling influence, especially upon muscular fibres."

We proceed next to the examination of certain processes, which are of extremely frequent occurrence in inflammatory exudations, or which impress a peculiar character on them, even in their nascent state. These are the suppurative, and that which gives rise to the granule cell and other forms of celloid corpuscles. We will first describe the products, and afterwards consider the mode of their production and their import. There are many varieties of *pus;* but that which is commonly called healthy (laudable) is that which we shall take for a typical description. It appears to the naked eye as a creamy, thick, opaque, and homogeneous fluid; communicates an unctuous feeling when rubbed between the fingers; is of a yellow or whity-yellow tint; sweetish, or insipid; and, while warm, gives off a peculiar, mawkish smell. Its specific gravity is 1030—1033. If allowed to stand some time in a tall, narrow glass, the fluid separates into a thickish *sediment*, more or less abundant, and a supernatant *serum*. This serum, according to Vogel (whose account we shall use freely) is identical with the serum of the blood, containing much albumen, extractive and saline matters, and fat. The reaction is alkaline; but it readily becomes acid, from the generation of an acid, which is commonly supposed to be the lactic. In some cases, however, according to Dr. Walshe, it has an acid reaction, even at the time of its formation. A peculiar substance, called pyin, by Guterboch, which Simon considers almost identical with mucin, is said by Rokitansky not to be one of the constituents of normal pus, but to proceed from croupous fibrin, in a state of diffluence mingled with it. The *sediment* consists almost entirely of small organized corpuscles, the well-known pus-globules. These are of spherical form, have a well-defined contour, formed by a distinct homogeneous envelop, inclosing a mass of soft granulous substance, and a varying number of nuclear corpuscles. These are, in well-formed pus-globules, for the most part concealed by the surrounding substance; but in the younger cells, even of healthy pus, and in all those of pus of an inferior kind, they are easily perceptible, even without the aid of acetic acid. Occasionally, a single nucleus exists; but more commonly it is made up of two, three, four, or even five large granules.

[1] One of the best instances of the pure serous exudation (serosity, as some call it), is the fluid which distends the ventricles of the brain in acute hydrocephalus—100 parts have been found to contain 1 of salt, .4 of animal matter, and 98.6 of water.

The single nuclei are always the largest, and indicate, as we conceive, the most perfect kind of development. The more numerous the nuclear corpuscles are, the smaller do they become, so that the opinion seems very probable, that the perfect nucleus is formed by a coalescence of the smaller corpuscles; and that the bipartite, tripartite condition, &c., is

Fig. 16.



Corpuscles from a pustule.

(a). Large granulous exudation globules.
(b). Pus-corpuscles.
(c). Nucleated fibres.
(d). Pus-corpuscles, their nuclei brought into view by acetic acid.
(a'). Granular exudation globules, their nuclei brought into view by the action of water.

an indication of imperfect development. The nucleus is seated on the envelop, or is parietal, as it is termed. Its diameter is about $\frac{1}{8000}$ in.; that of the entire pus-globule about $\frac{1}{3000}$ in. Single (complete) as well composite nuclei, are seen floating in the serum of pus; but they are not very numerous. We have also observed, as well as M. Lebert, small homogeneous, or faintly granular globules, about $\frac{1}{8000}$ in diameter. The observer just mentioned describes some globules which often occur, mingled with those of pus, but which differ from them in several particulars, being smaller, more transparent, and non-nucleated. These he terms "pyoid." There is generally a small quantity of diffused granular matter mingled with the pus-globules. This is more abundant, according to our observation, in pus of low and ill development. It is not to be confounded with the so-called elementary granules, which Rokitansky describes as originally discrete, and, subsequently grouping together, to constitute the nuclei. Dr. Walshe says, that the chemical composition of these granules is not always identical; that they are sometimes soluble in ether, and sometimes exhibit the reactions of a protein compound. The formation of the pus-globule does not appear to take place in one uniform manner. The nucleus is generally stated to be first formed by the grouping together of granules, which appear in a fluid blastema. Around this there may be first formed the envelop,

closely embracing the composite mass, so as only to be brought into view by the endosmotic action of water, or, as we think is more frequent, a granulous deposit forms round the nucleus, and afterwards becomes limited and inclosed by a cell wall. Lebert describes the pus-globules as being formed in a different manner, which Rokitansky also seems to admit, and which we think actually, though not constantly, occurs. According to this view, minute granular globules, forming in a fluid blastema, grow and enlarge, granules appear in their interior, and gradually assume the appearance of the composite nuclei. The globule is, in fact, from the first, a miniature of the fully developed one. It is formed, in the French phrase, "de toutes pièces." The recognition of this diversity in a formative process, is surely an important step. It is stated by Vogel that pus may be formed from a solid blastema of coagulated fibrin. "The pus-globules," he says, "are at first scantily dispersed through the stroma. Afterwards they become more abundant, and ultimately occupy the whole space, while the solid fibrin disappears." In this way, he states, pus is formed in all cases, where suppuration is consequent upon induration, as in the lung after hepatization, in solid exudations upon serous surfaces, and so on. In Rokitansky's opinion, these instances of suppuration really depend on the original combination of elements of pus with croupous fibrin. The latter breaks up, and becomes a diffluent mass; but yields nothing in the way of nutriment to the pus elements, which increase and develop themselves solely out of the sero-albuminous fluid, mingled with the fibrin. The changes taking place in intra-vascular coagula, which we have before described, bear upon this question, in connection with which we would also refer to some excellent remarks, by Dr. Walshe, in his article on Advent. Products. (*Cyclop. Anat. and Phys.*) To us it appears that it must be conceded, that elements may form in softening fibrin, indistinguishable from those of pus; and, indeed, Rokitansky's own description of croupous fibrin proves that such are present from the outset, so that we are inclined to adopt Vogel's opinion, that solid effusions may undergo actual conversion into pus, just as we know they may into fibroid tissue. The pus-globule is not very remarkably affected by being placed in water. It becomes somewhat swollen and more spherical, but is not destroyed and burst so rapidly as the blood-globule—the nucleus becomes somewhat more apparent. In blood, urine, mucus, saliva, it is unaltered. Acetic acid renders the granulous contents translucent, and brings out the nucleus more definitely. It renders the envelop also more transparent, but does not destroy it. Other dilute acids have a similar effect. Caustic and carbonated alkalies, and borax, convert the whole corpuscle into a viscid mass, leaving only very minute dark molecules, whose import is uncertain. Pus, Dr. Walshe says, possesses a remarkable power of resisting decomposition. At the end of months some corpuscles may still be found unchanged, among others that are dissolved. It even retards the putrefaction of substances which are placed in it; but at the same time seems to exert upon them a corrosive influence. Pieces of flesh, put into fresh pus, gradually lost weight, and were at last dissolved, without any evidence of putrefaction having occurred. Mr. H. Lee has recently shown, that pus possesses a remarkable power of accelerating the coagulation

of blood. In one experiment the blood, which had pus (healthy) added to it, coagulated in six minutes; while that which was left by itself re-

Fig. 17.

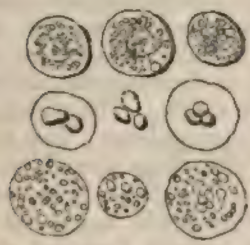


Fig. 18.

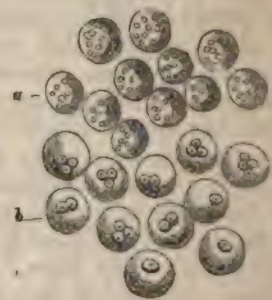


Fig. 19.

Fig. 20.

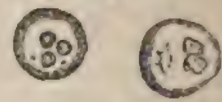


Fig. 21.

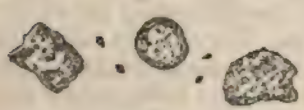


Fig. 22.

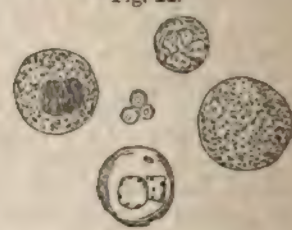


Fig. 23.

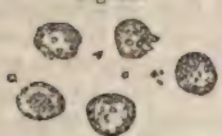


Fig. 18. *a*. Natural appearance of pus-corpuscles. *b*. Appearance after application of acetic acid.
Fig. 19. Pus-corpuscles, magnified 400 diameters.
Fig. 20. Healthy pus-cells.
Figs. 21, 22, 23. Various forms of pus-cell from phlebitis and pyæmia.

quired twelve. The pus must, of course, act upon the fibrin; but of the nature of the change, we have no knowledge. The following analysis, by Dr. Wright, exhibits the main features of the chemical constitution of pus very well. They apply, of course, to pus as a whole, not to the serum only. The large quantity of fat in pus is remarkable, as well as the amount of albumen—the latter sometimes exceeding that contained in the liquor sanguinis. This is probably to be explained by the dissolution of some of the red globules, and the blending of their albuminous globulin with the exudation that yields the pus.

	From a vomica.	From Psoas abscess.	From Mammary abscess.
Water	894.4	885.2	879.4
Fatty matter	17.5	28.8	26.5
Cholesterin	5.4		
Mucus	11.2	6.1	
Albumen	68.5	63.7	83.6
Lactates, carbonates, sulphates, and phosphates of soda, potash and lime	9.7	18.5	8.9
Iron	a trace		
Loss	3.3	2.7	1.6

In various unhealthy states of the system pus is formed, which differs in several respects from that which we have now described. Mucin, the peculiar principle of mucus, may be more or less abundantly dissolved in the serum, which may be recognized by the coagulation produced by acetic acid and alum. Small fibrinous flakes, epithelial particles, cholesterin scales, and prisms of triple phosphate may also be mingled with it, as well as varying quantities of free oil. The pus-globules are in such cases often ill-shaped, feebly formed, conveying the idea of very defective formative power; the quantity of granular matter mingled with them is much increased.

Fig. 24.

One variety of pus has been called *ichor*, and is especially distinguished by the paucity of its corpuscles, which, indeed, Vogel says, are absent when it is perfectly pure. Its color is reddish, or brownish red, it is alkaline, and contains a considerable quantity of albumen. Its presence indicates an exceedingly depressed state of the vital formative power. Certain unhealthy kinds of pus, which Rokitansky comprises under the term (Jauche) sanies, are especially distinguished by their corrosive action upon the tissues, which he contrasts particularly with the bland quality of healthy pus. Their appearance is not at all constantly different from that of the normal fluid, but they are apt to be thinner, more tinged by hæmatin, of an offensive, or ammoniacal smell, and to communicate a sensation of pricking or itching to the finger when applied to them. Their corpuscles are stunted, and their developments are variously altered, apparently by the "*gnawing*" action of the serum in which they float. It seems to us very questionable, whether the dissolving action which pus is said to exert on pieces of dead flesh, belongs at all to it in a healthy and fresh state, and whether it does not really depend on the generation of acids within itself, in consequence of decomposition. Purulent effusions may degenerate into a semi-fluid amorphous mass, the corpuscles breaking up, and the serum undergoing chemical changes, often of a putrefactive kind. They may also undergo fatty degeneration, calcareous salts being liberated, or deposited at the same time. Either of these two changes being premised, it is possible that a purulent collection may be absorbed, but it is only too probable that in the former case the result will be a fatal contamination of the blood by the decomposing matters taken up into it. Apparent temporary absorption may be easily produced by means which, creating a considerable demand for fluid, withdraw the serum

from the pus-corpuscles; but as these retain their vitality, they soon attract a fresh quantity of blastema from the blood, and the abscess remains undiminished. This persistent vitality of the organized corpuscles of a fluid which is regarded as effete in the highest degree, and incapable of any further development, is certainly remarkable; one would rather have expected that they would have disintegrated rapidly. Pus may be confounded with some other fluid, and the distinction is sometimes only to be made out by careful microscopical examination. What we have said respecting softened fibrin, will show that a fluid having this origin may approach very closely to the purulent product of inflammation; so much so, that it may be doubted whether it be not in part identical with it. It has happened several times, that a quantity of desquamated epithelium, the particles being partly entire, partly broken up, has been mistaken for a collection of pus. Vogel records an instance of this in the pelvis of the kidney of a person who died with empyema. The pus-like fluid accumulated in the urinary passages, was deemed convincing proof that absorption of the thoracic effusion had been taking place with subsequent elimination of the same by the kidneys. The microscope, however, showed that the whitish-yellow, thick, creamy fluid which had been considered as pus by all who saw it, consisted entirely of epithelial debris. We shall presently describe mucus, and will then point out wherein we believe it to differ from pus. A caution is necessary against a very possible error which even practised observers have committed, viz: that of mistaking the colorless corpuscles of the blood for pus-globules. The two bodies are very much alike, but the blood-corpuscle is somewhat smaller, generally more finely granular, and with rather less definite contour. The interior nuclei of. both are identical.

The granule-cell, exudation globule, or glomerulus, which was first described by Gluge, and called by him the compound inflammatory

Fig. 25.

Glomeruli and granulous cells.

(1) From ovarian cyst.
(2) From cancer of breast.
(3) From inflamed lung.
(4) From inflamed pia mater.
(5) From a case of tuberculous meningitis.

The opaque cells are the glomeruli, the more simply granulous are the granular cells.

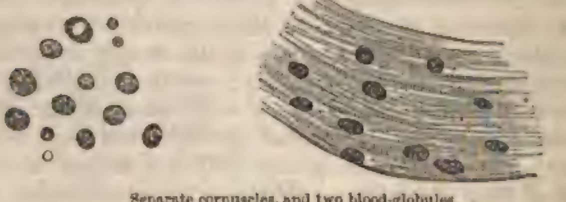
globule, is very frequently present in exudation, and is, speaking generally, a valuable sign of the existence of the inflammatory process, but

not an infallible one. The granule-cell is usually of large size, from $\frac{1}{3400}$—$\frac{1}{500}$ inch, mostly spherical, but often oblong, or of irregular shape. By transmitted light they appear dark, on account of their opacity; by direct, of a dead white. Their structure will be best understood from the account of their development. Our own observation exactly accords with that of Vogel, who states that there are first formed in the blastema nucleated granular cells, which gradually fill themselves with the peculiar opaque glistening granules characteristic of these corpuscles, until at last the nucleus is entirely obscured, and the originally smooth-cell membrane becomes rugged, the granular cell appearing as an agglomeration of granules. Subsequently the cell-wall vanishes, the granules separate from each other, probably on account of the dissolution of the uniting substance, and the corpuscle breaks up into a loose heap of oily-looking granules. Vogel says that caustic potash and ether sometimes, but not always, dissolve these granules; Rokitansky regards them as of fatty nature, and considers the process of granule-cell formation as one of fatty degeneration of pre-existing cells. He says that granule-cells do not form in blastemata devoid of cells, and that any cell may undergo this transformation, a cancer-cell, a pus-cell, as well as the cells that form in exudations. There is no doubt that blastemal exudations, devoid of cells, often break up into collections of the fatty-looking granules, and that they may assume this form from a very early period; as, for instance, in the coating of the vessels in the gray matter in meningitis. Corpuscles also, quite indistinguishable from granule-cells, occur in lungs that bear no trace of having ever been inflamed, and this in considerable numbers. We do not think, therefore, that Rokitansky's view of the nature of the process is quite correct, but are inclined to believe that, generally, where blastemal exudation has been poured out in greater quantity than is necessary for the nutrition of the tissue, it *may* undergo such a change, as that its oil, in combination with a part of its albumen, separates in the form of glistening granules, while the remainder undergoes absorption, or is otherwise consumed. The granules appear to be attracted towards the interior of existing cells; we have distinctly seen them coating the outside of a cell. One of the causes of the different size of the granule-cell depends on the circumstance that cells of very different magnitude, and cells in very different stages of growth, may be the seat of their deposition. Between the suppurating process and that which forms granule-cells, a wide separation exists; the former, as we have seen, gives rise to a fluid essentially effete, rarely, with difficulty, or with peril, capable of being absorbed; the latter involves no such deteriorating alteration of the blastema; its occurrence, on the contrary, is eminently favorable to reabsorption of an exuded mass. It is to be remarked, in conclusion, that a very abundant cell-growth commonly takes place in exudations, many particles of which are correctly denominated granular cells. These are not to be mistaken in descriptions for the *granule-cells*, into which they often undergo metamorphosis. The similarity of the name is unfortunate, but the subjoined sketch will make the distinction between the two very apparent.

The last inflammatory product which we have to notice is mucus.

Speaking correctly, it is only unhealthy mucus which comes under this head; for it is perfectly clear that several internal membranes secrete a mucous fluid. The distinction between this and the morbid product is tolerably precise, and easy to be ascertained. The former is a tenacious, clear fluid, containing only some admixture of the epithelium of the membrane producing it, and having no special corpuscles of its own. The latter is loaded with corpuscles, identical with those of pus, together with a varying quantity of epithelial debris. Between such mucus and pus it is evident that a close analogy subsists. Mucus may be distinguished, like pus, into a fluid, the *liquor muci*, and corpuscles. The liquor muci, as we find it in the secretion of a membrane which has been subjected to moderate irritation, is a transparent, tenacious, more or less stringy fluid, of alkaline reaction, and more or less saline taste. The addition of acetic acid, or any weak acid, produces a kind of coagulation, and the formation of a granular precipitate, which Simon states is the mucine, the principal constituent of the fluid. This is held in solution by means of an alkali, and consequently falls on the latter being taken up by an acid. Not much is known of this substance, except that it is a protein compound. Albumen or fibrin, treated with liquor potassæ, forms a transparent, viscous mass, having much resemblance to its solution. The proper corpuscles of morbid mucus are, as we are fully persuaded, and as the best observers state, quite identical with those of pus. They are usually mingled with epithelial particles, in very various stages of their formation, from a simple nucleus up to a complete cell. It is only in cases of prolonged and rather intense inflammation that traces of epithelium are wanting, and the so-called mucous corpuscles are crowded together, and seem to load the fluid. In mucus expectorated by persons of very depressed powers, the cor-

Fig. 26.



Separate corpuscles, and two blood-globules.

puscles may be seen feebly formed, like those of pus secreted under similar circumstances; the granular contents of the cell are deficient, and allow the composite nuclei to be distinctly seen. It is often very observable how the tenacious fluid, in which the corpuscles are entangled, in consequence of being dragged in one direction, produces an alteration of their shape; they thus become oval, or even staff-shaped. Granulous and oily matter is commonly diffused through the liquor muci, just as it is through that of pus. It is manifest, from what has been stated, that the difference between mucus and pus consists essentially in the different nature of the fluids, not in that of their corpuscles. Both are exudations; but the one is poured out directly from the bloodvessels, as

an albumino-fibrinous blastema, in which special corpuscles (the pus-cells) are formed; the other transudes, not only through the capillary walls, but through the basement-membrane of the mucous surface, with more or less of attached epithelium, and in so doing experiences a peculiar modification, which remains impressed upon it, while the corpuscles mingled with it are either the natural cell-growth of the surface, or such as form naturally in blastemata, that are destined to become effete. Mucus, it is evident, is effete, like pus. It is hardly possible that any part of it should be absorbed again. A constant flow of it becomes, therefore, a serious drain upon the system, entailing a loss of so much protein matter. The old question, as to the means of distinguishing between pus and mucus, is manifestly of little moment, and has, in general, no interest for the practical physician. It is sufficient to state that the liquor puris is albuminous, the liquor muci not so; that pus will mix with water, and mucus will not; that pus is dissolved, in some measure, by acetic acid, while mucus is coagulated; and that mucus generally contains traces of epithelium, while pus does not. It may, however, be observed, that if a fluid, secreted under inflammatory irritation, should lose the characteristic tenacity of the liquor muci, and come to contain albumen, there would be considerable reason to fear that the texture of the mucous membrane had become ulcerated, and that the albuminous exudation, no longer modified to mucus, was being poured out from exposed vessels. Any admixture of blood with the secretion would render this still more probable.

Having considered the effusions of inflammation, we come next to examine the various changes that may take place in a part inflamed. We enumerate these as: (1) Enlargement; (2) Atrophy; (3) Ulceration; (4) Gangrene. The term "enlargement" is preferable to that of "hypertrophy," which is sometimes employed, because it conveys no such erroneous idea as that the part is truly increased in size by addition of more of its own proper substance, an occurrence which most rarely, if ever, is the result of any form of inflammation. The enlargement depends entirely on the infiltration of the tissue with some form of exudation matter, which subsequently undergoes metamorphoses such as we have described, and is more or less completely absorbed. It often happens, however, that a part remains behind, and is converted into a low form of fibroid tissue, or a semi-solid blastema, imbedding multitudes of nuclear particles. This constitutes induration-matter, which resembles very much that which forms cicatrices; like which, its tendency is to contract and shrink, thus compressing and obliterating the vessels of the part, and in this way, as well as by its pressure, inducing the atrophy of the tissues among which it is deposited. A good instance of primary enlargement and subsequent atrophy, resulting from inflammation, is afforded by some cases of cirrhosis of the liver. Rokitansky describes atrophy, the result of inflammation, as depending upon the mechanical injury done to the tissues, in the seat of inflammation, by the exudation, as well as upon their being deprived by it of their proper amount of nutrition. Being thus rendered unfit for the discharge of their function, they fall to pieces, and are absorbed, together with the exudation. "This occurs with especial frequency in delicate, lacerable tissues, when

large quantities of exudation have been effused, and such as are solid and capable only of slow reabsorption. Thus, in the inflammatory foci, the substance of the brain, of the muscles, of the kidneys, &c. becomes lost, while there remains in its place one or more gaps, limited by cicatrix-tissue, which, if such gaps are small and numerous, causes a spongy, rarefied condition of the tissue."

Ulceration implies that condition of a part in which more or less of its proper substance has become eroded, and has disappeared, in consequence of unhealthy action, so that a cavity remains. This condition does not most commonly exist alone, but together with a greater or less amount of exudative and organizing processes. These are so far from being essential to it, that they constitute, in fact, the means by which its ravages are repaired; the formation of granulations, and the effusions of pus, are the characters, not of an extending, but of a healing ulcer. Instances of pure and simple ulceration are to be seen in the cornea, and in some ulcers of the walls of the stomach; they penetrate the tissue more or less deeply, so as sometimes to perforate it, without any surrounding thickening from the deposition of lymph. When the erosion of the tissue goes on rapidly and extensively, forming a sore, with very irregular surfaces and margins, and presenting no trace of reparative action, the ulceration is said to be phagedenic. Many other varieties of ulcers are mentioned, but they all have reference to the amount and character of the exudative and reparative processes taking place; and though they afford excellent indications of the condition of the general system, which are well worth studying, they are not to be regarded as containing anything special in the nature of the ulceration itself. Rokitansky considers that the main circumstance determining ulceration, is the corrosive quality of the exudation, the ichor. We agree with Mr. Paget in doubting the correctness of this as a general statement; it is much more probable that, in consequence of altered and defective nutrition, the tissue gradually deliquesces (so to speak) into a fluid, returning thus, though spoiled and effete, to the form of the healthy blastema, from which it originated. It is matter of some dispute whether the tissue, as it decays and is destroyed, is removed by absorption, or is cast off from the broken surface. Mr. Paget inclines to the opinion that it is ejected, resting upon the analogy of excreting surfaces, on the discovery of fragments of bone and phosphate of lime in ulcers of osseous structures, and on direct observation of the commencement of ulcers. We are also inclined to think that the process of removal is rather by ejection than by absorption, especially in the case of open ulcers, yet so that some amount of absorption also takes place, varying in degree in different cases, and probably even predominating in those where there is no external outlet. The formation of ulcerations on the surface of the cervix uteri, has appeared to us to take place in the following way, much as it is described by Dr. Baly on the intestinal surface: As the first step, in the situation of a spot of hyperæmia, a minute vesicle is formed, the epithelial layer being lifted up by effused fluid, while the tissue beneath is softened, loosened up, and appears less dense than natural. Afterwards the covering of the vesicle is detached, the fluid escapes, and the tissue beneath appears still more lax and

spongy, and has evidently undergone loss of substance. The hyperæmia, persists. In this case, we feel little doubt that the deliquescing tissue is partly thrown off in the fluid which escapes from the vesicle, partly absorbed by the bloodvessels.

It seems desirable to indicate the difference which exists between ulceration and absorption. In both, there may be considerable loss of substance at some one or more points of the part affected, but in ulceration there is always an unhealthy state of the nutrition of the tissue, there is disease of it; in absorption, this is not the case: the part may be diminished, but cannot be said to be diseased. Contrast a bone, carious and ulcerated from inflammation, with one which has undergone absorption, in consequence of the pressure of an aneurism.

The last result of inflammation which we have to mention is Gangrene, or Mortification. This, indeed, is not a very common termination, nor is it at all peculiar to the inflammatory process. It more really belongs to a deficient condition of vital power induced by various causes, which may of itself be the cause of the death of some part, or render it so feeble that it perishes under injurious influences which would otherwise have had no such effect. Gangrene may ensue from the following causes: (1) from an absolute and prolonged stagnation of the blood; (2) from a defective supply of blood; (3) from a general taint or unhealthy crasis of the mass of the blood; (4) from a local injury. The absolute stagnation of the blood in the first case may be the result of violent inflammation, especially of an asthenic kind, and occurring in debilitated systems and organs; or it may be brought about mechanically, as when a portion of intestine is strangulated. Rokitansky says, that in this case the blood stagnant in the vessels *first* undergoes gangrenous decomposition, and that, exuding through their walls in the state of gangrenous ichor, it sets up the same decomposing change in the surrounding tissues, which break up into a dark-colored pulp, of as little consistence as tinder; diffluent, and excessively stinking. In the second case, besides various kinds of obstruction of the arteries from external pressure, their channels may be blocked up by extensive fibrinous coagula, either forming spontaneously, or in consequence of disease of the coats of the vessels. Gangrene occurring in aged persons, without any apparent cause, that from the use of diseased grain, and hospital gangrene, are instances in which the morbid action is dependent on a general taint of the blood, or decay of the whole system. Mr. Simon suggests that the mode in which ergot of rye produces its fatal effect, may be by causing such contraction of the bloodvessels as prevents the flow of blood into the more distant parts, which consequently fall into the condition of dry gangrene. Spontaneous gangrene in old persons, or others, in which after death no obstruction of the bloodvessels is found, can only depend on an actual and premature loss of vitality in the part affected, the tissues of which are no longer able to carry on the actions of vital chemistry, and yield to those of inorganic, *i. e.* decompose, before the death of the system has actually occurred. In gangrene, from local violence, or from frost-bite, &c., the vitality of the tissues of the part is destroyed by the injury done to them. The general characteristic of gangrene in all these cases is the failure of

vital action; decay and death in the tissues, intense inflammation, absence of blood-supply, a poison circulating in its current, senile decrepitude, a fearful laceration, may all have the effect of dissolving the vital affinities which hold together the elements composing the complex substances of our organism, and allowing them to fall back, as they naturally do, into the simpler compounds of inorganic chemistry. The distinctions of dry and moist gangrene, of black and white, of inflammatory and cold, have reference very much to the state of the affected part, with regard to the supply of blood. If the gangrene have its origin in inflammation, there will be a considerable quantity of fluid ichor effused, and the color of the part will be of a deep red, or almost black. On the contrary, if the gangrene depend on deprivation of the supply of blood, the part will be more dry, and of a pale color. Sometimes, especially from the effect of ergot of rye, a limb dries and shrinks up, becomes mummified, as it is said, with little change in color. A black color is, however, often observed in parts affected by *gangræna senilis;* this, no doubt, depends on alteration of the blood in the vessels, though there is often no hyperæmia. Soft tissues are more liable to mortify than such as are of a firmer consistence; bones, elastic and fibrous tissues, resist longer than muscles and mucous membranes; the large vessels and nerves are sometimes seen completely exposed by the ravages of hospital gangrene, all the tissues being removed from around them.

The constitutional disturbance which often supervenes on gangrene is easily to be accounted for by the absorption of decomposing matters into the blood, which act as a virus upon it, and render it unfit to maintain healthy action.

PYÆMIA.

Proceeding to consider various diseased states of the blood, we come next to one, in which a product of inflammation, viz: pus, is believed in some way to be mingled with the blood, and, by poisoning it, to produce both general fatal depression of the powers of life and local purulent accumulations, the so-called *secondary depots*, in various important organs. The phenomena observed in pyæmia are somewhat as follows: A man has received an injury, or undergone some surgical operation, it may be an amputation, or that for fistula in ano; for a time all proceeds well, but soon shiverings come on, with adynamic fever and oppression, he emaciates, pain or disorder shows itself in some internal viscus, and in a few days he dies in a state of stupor, or delirium. On opening the body, the blood is found less coagulated than is natural; there are abscesses more or less numerous commonly in the liver and lungs, and often in other parts; there is frequently purulent effusion in the cavities of the joints, and sanguineous or purulent effusion in the serous cavities also. The question to determine is, how these morbid changes are brought about. From the almost invariable occurrence of such phenomena in persons who were the subject of suppuration, or in whom it was reasonable to believe that pus might be formed in the seat of some

injury, it was natural to conclude that the pus, making its way into the blood, was the cause of the mischief. This was confirmed by Cruveilhier's experiments of injecting mercury into a vein, after which there was found in the centre of each of the small abscesses a globule of the metal which thus seemed to have been carried in the circulation to the part where it was deposited, and where it excited inflammation passing into suppuration about itself. The pus-globule was supposed to act in the same way as the globule of mercury; being too large to traverse the capillary channels it was arrested there; and similar obstructions taking place in other points of the same organ, a number of separate inflammations, which soon suppurated, and formed the so-called multiple abscesses, were thus established. Another confirmatory fact of the same view was observed in these experiments, viz: that the mercury was arrested almost entirely in the first set of capillaries at which it arrived; if it was injected into tributaries of the portal vein, the abscesses were found in the liver; if into veins of the general system, the abscesses were in the lungs. Pyæmia follows the same law, however, less closely; it is very common to find numerous abscesses in the liver when pus can only have been conveyed from the veins of the general system: on the other hand, that which is carried by the portal vein to the liver often seems to be entirely arrested there. There is no doubt that it is the presence of puriform matter in the blood which gives rise to the phenomena we are considering, but it is not yet fully ascertained whether perfectly-formed pus circulates in the blood, or only a pyogenic fluid, nor how either of these is introduced within the vessels. Before we enter further on these points, we will describe more particularly the formation of the multiple abscesses. M. Lebert, whose observations accord very closely with our own, notices particularly that the parts which are the seat of purulent effusion are truly inflamed; parenchymata, synovial, or serous membranes, if examined at all at an early period, are found in a marked state of inflammatory hyperæmia. This proves that the term secondary depots, sometimes used, is incorrect; the pus is actually generated, not only deposited in the part. The stages of the forming abscess are as follows: "(1.) A local and circumscribed capillary injection, showing little vessels dilated and gorged with a dark red blood, more or less coagulated, in which are seen few globules, and very uniform plasma, but never pus-corpuscles. (2.) In this centre of the vascularization a yellow point begins to be seen, which is nothing more than a drop of pus." The microscope shows in it some well-marked pus-globules, generally without nuclei, and especially many granules, all floating in a pyoblastic serum. (3.) The vascularity declines, the purulent collection increases, infiltrating the tissue, the elements of which are not destroyed. (4.) The secretion of pus continues, and the purulent inflammation is transformed into an abscess, bounded by a margin of red injection, and having its interior, in the case of the larger ones, lined by a soft pyogenic membrane. The larger abscesses have commonly a very irregular form, which results from the fusion of several smaller ones together, as they go on increasing in size. As showing the truly inflammatory character of the pus-secreting process, we may mention that we have found the texture of the cartilage of the knee-joint

altered just as it is in common arthritis with ulceration after death from pyæmia of only a few days' duration. The cartilage in the case referred to was ulcerated, and the joint contained pus. M. Lebert has made several careful observations, with a view to discover whether the pus-globules are actually present in the blood of the pyæmic; the result of these seems to be, that they cannot certainly be detected; even in the blood of animals that died from the effects of pus injected into their veins, the globules of pus could only once be discovered; and it seems incontestable, that in the great majority of cases they are rapidly destroyed after having entered the circulation. This throws considerable doubt on the view above noticed, that the pus-globules become arrested in the capillaries, in consequence of their size, and thus establish numerous foci of inflammation. Rokitansky also expresses his opinion very strongly against it. He considers that pyæmia occurs not uncommonly as a primitive affection; that is to say, that pus is actually formed by and in the blood itself, in consequence of certain changes in the fibrin, such as occur in the croupous crasis, which we shall afterwards notice. This supposition would account for cases occasionally met with, in which there are multiple abscesses, yet no source of purulent infection can be discovered. The more common case of consecutive pyæmia, he states, may originate in either of the three following ways. (a.) By the absorption of the *serum* of pus, either into the lymphatics, or into the bloodvessels directly. (b.) By the reception of pus into bloodvessels which have been in any way opened, especially into those which traverse solid formations in which the mouths of the vessels are likely to be held open. (c.) Particularly by the flowing off towards the veins of pus, which has been produced in a local process (capillary phlebitis) within the vessels. We think it important to recognize the possibility of pyæmia taking place from absorption of serum alone, because it is clear that this may easily take place wherever capillary vessels are in contact with puriform exudation. The reason why it does not more often occur is, we conceive, that in a tolerably healthy state the fluid absorbed from the pus is not adequate to contaminate the mass of the blood; it undergoes certain chemical changes, and is soon eliminated as effete matter. But in a depressed state of the vital powers, the blood cannot resist and throw off the contaminating matter, and a pyogenic diathesis is established. This opinion seems to be confirmed by the experiments which have been performed upon animals. Lebert found that rabbits which had serum of pus injected into their veins, did not survive much longer than those in which pus was injected entire, but that dogs showed no serious morbid symptom after the injection of serum. In the experiment performed by Mr. H. Lee, we find that dogs and asses recovered from the effects of the injection of pus, or at least were recovering when the injection was repeated. All this shows that the weaker the system, the less it is able to resist the poisonous effects of pus, or its constituent parts; and should lead us to guard, as far as possible, against the supervention of pyæmia after operations on debilitated patients. The third mode by which pus comes to be mingled with the blood is undoubtedly the most common, and is believed by Lebert to be almost the sole one. He remarks that, in the great majority of cases, some trace of inflam-

mation of the veins may be discovered, and supposes that where they cannot, the veins affected are so small, or so obscured, that the source of the disease is overlooked. We believe that in such cases, pyæmia originates in either of the two other modes. The injection of pus into the living blood tends, as Lebert states, to diminish its fibrin, to destroy its globules, to alter its normal cohesion, and to precipitate a part of its fatty principles. The blood thus altered tends to form ecchymoses, capillary hemorrhages, especially in the lobules of the lungs. Rokitansky describes the bodies of those dead with pyæmia, as presenting only a brief rigor mortis, a lax and pale condition of the muscles, especially discoloration and lacerability of that of the heart, rapidly advancing putrefaction, with extensive stains from exudation. The lungs are especially the seat of dark hypostatic congestion. The coagula of the blood in the heart and large trunks are small and soft, and the inner membrane of the vessels stained. It is very intelligible how blood thus vitiated should tend to stagnate at various points of the capillary plexuses which it traverses, and to form there minute abscesses, without supposing that the vessels are actually blocked up by entire pus-globules. The obstruction is of a chemico-vital, not of a mechanical origin.[1] The contamination of the blood when effected, is clearly of such a nature, that it determines the rapid suppuration of all exudations; there is a strong tendency to the formation of pus, a true pyogenic diathesis. Were it not for this, the formation of the multiple abscesses would not take place so quickly. The fact stated by M. Lebert is interesting, that muco-pus, the product of an inflamed mucous membrane, produces, when injected into the blood, the same effect as pus, a further proof of the identity of the corpuscles of the two fluids.

LEUCOCYTHÆMIA—LEUKHÆMIA.

These terms—the former signifying white cell-blood, the latter, simply white blood—have been given to a condition of the circulating fluid, which is not of very common occurrence, and has only been recently discovered. The former name is the more correct, as the blood does not lose its red color, and as the prominent alteration in it is the great increase of corpuscles, resembling the white or colorless ones naturally present. When a drop of blood, drawn during life, is examined microscopically, the red corpuscles appear tolerably natural,

[1] In the following case, the formation of the secondary depots seems to have depended on a coagulation of the blood in certain spots, with simultaneous exudation of fibrin. A girl, aged nineteen, died after nine days' illness, having been previously in perfect health. There was purulent matter in and around several of the articulations. The lungs contained several masses, mostly about the size of a pea, and situated near the surface. Some were dark red, well-defined, and exhibited under the microscope fibrin in strands, together with numerous blood-globules, granular cells, and celloid particles. Others appeared as whitish defined masses, showing scarce any trace of fibrinous coagulum, but multitudes of well-shaped nuclei, and celloid particles, not resembling pus-globules, with some oily and granular matter. In others, again, the central part was softening down, while the outer remained firm, the central softened part never contained pus, but granular and oily matter with varying quantities of corpuscles. The most advanced presented a capsule of firm grayish indurated matter, with soft broken up contents.

and often arranged in rouleaux, leaving intermediate spaces, which are more or less crowded with the white corpuscles. It is difficult to say what is the proportion which the one set of corpuscles bear to the other. In the case which we witnessed, we should have regarded them as nearly equal, comparing mass to mass. Dr. Bennett estimates the

Fig. 27.

Blood in Leucocythæmia—four of the white corpuscles have been treated with acetic acid. From Dr. H. Bennett's work.

white as scarcely one-third the number of the red. Many of the white corpuscles are very much larger than the natural size. They have more coarsely granular contents than the normal ones, with an interior single, double, or tripartite nucleus. The envelop and nucleus are brought into view distinctly by the action of acetic acid, which renders the granular contents transparent. Occasionally, a crescentic nucleus is to be seen in the cells, and some free nuclei are also observed between them. The blood, in fatal cases, is often found imperfectly coagulated—sometimes grumous, of a dirty brown color. The coagula, where decolorized, have not the aspect of healthy fibrin, but are of a more opaque dull yellow; and, when broken up, resemble thick creamy pus. They contain in this part very numerous white corpuscles, to which the peculiar aspect is probably due. In the case where the blood has been analyzed, the fibrin exceeded the normal amount. Perhaps this increase may be rather apparent than real, in consequence of numerous white corpuscles being included in the fibrin. The red corpuscles are invariably diminished; the solids of the serum little altered. Morbid changes are chiefly observed in the spleen, the liver, and the lymphatic glands. The spleen is often very greatly enlarged, apparently by a kind of true hypertrophy of its nuclear structure. It has, however, been found healthy. The liver is less frequently enlarged. It was so in about half the number of cases. Its texture is more or less altered. The lymphatic glands seem to have been enlarged or cancerously diseased in eleven out of nineteen cases. The affection has been more often observed in males than females, in the ratio of 16 : 9. It seems "to be most common in adult life, and more frequent in advanced age than in youth." The respiration is often interfered with by the distension of the abdomen. Diarrhœa is a frequent symptom, vomiting is less often present. Hemorrhage, from various parts, was observed in the majority of cases, and was attended with purpura hemorrhagica in one instance. In about half the number of cases observed, dropsy was present, generally dependent on the abdominal tumors. Some febrile disturbance is not unfrequent, but not

to any great degree, or of long continuance. Anæmia is commonly well marked, and emaciation, in the fatal cases, is said to be extreme. There seems no reason to believe that the affection is at all connected with ague, or the malarious poison. The foregoing facts, relative to Leucocythæmia, we have taken from the memoir of Dr. Hughes Bennett, who offers the following theory of its nature: He regards the spleen, thyroid, supra-renal, pituitary, pineal, thymus, and lymphatic glands, as constituting a great glandular system, whose office it is to form the blood-corpuscles. These are for the most part thrown off from the organs mentioned, and enter the circulation as colorless nuclei, identical with the peculiar corpuscles of these glands. Sometimes, however, the nuclei proceed to cell development, and appear then as the "white corpuscles." The nuclei of these multiply by a process of division, circulate in the blood with colorless cells, and subsequently escape and become colored blood-globules. Now, "in certain hypertrophies of the lymphatic glands," Dr. Bennett believes, that "their cell-elements are multiplied to an unusual extent, and under such circumstances find their way into the blood, and constitute an increase in the number of its colorless cells. This is leucocythæmia." Our limits forbid discussion; and we can only say, that all our observations respecting the development of the red globules are entirely opposed to the view maintained by Dr. Bennett; and that we are fully convinced that, whatever action the glands referred to exert upon the fluid part of the blood, they furnish none of its corpuscular elements. The blood, we believe, forms its own floating cells, and these may, of themselves, become diseased and variously altered. Neither can we admit that the external similarity of the white granular cells in leucocythæmia to the natural white corpuscles, is a certain proof of the one being merely further developments of the other. Cells of similar aspect may have the most different properties. No other conclusions can be formed at present, as we think, than that leucocythæmia is a peculiar blood disease, whose cause and mode of origin is quite unknown.

NECRÆMIA.

This term is applied by Dr. Williams to that condition of the blood, in which it appears to be itself primarily and specially affected, and to lose its vital properties. It is, in fact, "death beginning with the blood." "The appearance of petechiæ and vibices on the external surface, the occurrence of more extensive hemorrhages in internal parts, the general fluidity of the blood, and frequently its unusually dark or otherwise altered aspect, its poisonous properties, as exhibited in its deleterious operation on other animals, and its proneness to pass into decomposition, point out the blood as the first seat of disorder; and, by the failure of its natural properties and functions, as the vivifier of all structure and function, it is plainly the medium by which death begins in the body." . . . . . "The blood, the natural source of life to the whole body, is itself dead, and spreads death, instead of life. The heart's action is faltering and feeble; the atonic vessels become the

seat of congestions, and readily permit extravasations. The brain, insufficiently stimulated after slight delirium, lapses into stupor; the medulla no longer regularly responds to the "besoin de respirer," and the respiratory movements become irregular. Muscular strength is utterly lost; offensive colliquative diarrhœa, or passive intestinal hemorrhage, often occurs; sloughy sores, or actual gangrene of various parts is very easily produced; and putrefaction commences almost as soon as ever life is extinct. The track of the superficial veins is marked by bloody stains; hypostatic congestion takes place to a great extent; the blood remains fluid, and stains the lining membrane of the vessels. Rokitansky describes the blood as often foamy, from the development of gas—of a dirty red raspberry-jelly color; its serum dark, from exuded hæmatin; and its globules swollen up by endosmosis. Coagula are either totally absent or are very soft and small. The exudations are of a dirty red—turbid, thin. There is scarce any rigor mortis; the tissue of the heart and of other organs is flaccid and softened, and stained by imbibition of the serum. Gas is quickly formed in the vessels and in the areolar tissue, giving rise to a kind of emphysema. It is very remarkable that this necræmic condition, or one closely resembling it, may be brought on by violent shocks inflicted on the nervous system, as well as by the introduction of miasmata or animal poisons into the circulation. Violent convulsions, overwhelming emotions, the shock of an amputation, a stroke of lightning, even a severe exhausting labor, are mentioned by the German pathologist as having produced this effect. More common causes, however, are malignant scarlatina and typhus, yellow fever, the plague, and the disease called glanders. It may be said, generally, that the *early* appearance of sinking and prostration in any fever indicates that the blood is thus seriously affected. We are ignorant what is the exact nature of the changes which take place in this condition of the blood. Probably they are more of a vital than merely chemical kind—that is, they affect the properties of the blood more than its composition. The blood-globules do not appear to be destroyed, but they circulate probably some time before death, as so many dead particles prone to be enlarged and to stagnate in the capillaries, and to part with their contained hæmatin. The fibrin is in great part destroyed; but how this comes to pass we are ignorant. We can perceive, on the whole, scarce anything more than that the powers of vital chemistry rapidly decay, and those of ordinary chemical affinity usurp their place.

CRASES OF THE BLOOD.

There are yet several morbid conditions of the blood which are scarce recognized sufficiently, at least among British Pathologists. These, Rokitansky describes as so many crases or alterations of the natural composition or *mixture* of the blood. They are often chronic, coming on imperceptibly, and, perhaps, scarcely noticed, until disorder begins to manifest itself in some particular organ; in other instances, probably as numerous, their development and manifestations take place rapidly,

and give rise to acute affections. There is very much reason to believe that they originate most of the serious visceral diseases which are so common. Very many cases of granular degeneration of the kidney, of cirrhosis of the liver, of contracted orifices of the heart, proceed, in our opinion, from slow and gradual textural changes, dependent on an unhealthy crasis of the blood. The importance of being aware of this, in the treatment of these affections, is abundantly manifest. As, however, our knowledge of these conditions of the blood is yet very imperfect, we shall not attempt more than to indicate shortly the principal features of the several crases, as they are enumerated by Rokitansky: A crasis may occur as the *primitive* affection, and its local manifestation, when it takes place, be determined, as to its seat, either by external influences, or by the operation of the nervous system. Or it may be *consecutive*, arising as the consequence of a local, morbid process, which has caused infection of the general mass of the blood by matter *absorbed*, or having undergone a deteriorating change *within* the vessels. A crasis may terminate, either by return to the healthy condition, or by conversion into another morbid crasis, or by destruction of life.

The *fibrinous crasis* corresponds to the condition of blood, which may be termed phlogistic or inflammatory. It is characterized by an increased tendency of the fibrin to coagulation, and to separation in a solid form, either in some part of the vascular system, or as an exudation in some of the tissues. For the development of this crasis, Rokitansky considers it necessary that the respiratory function should be freely performed. In most cases, the quantity of fibrin in the blood is much increased; but this is not so essential a feature as the alteration of its quality. In the croupous variety of this crasis both the coagula and the exudations show less tendency to organization; on the contrary, they tend to break up themselves, and often corrode, and, as it were, fuse down the tissues in which they are deposited. The mucous surface of the respiratory and digestive canals, serous and synovial membranes, are the chief seats of such exudations. The croup of early life, many pneumonias, many cases of puerperal peritonitis or phlebitis, acute rheumatism, and endocarditis, are so many examples of disease intimately connected with this crasis. Fibrinous crases often appear epidemically. It may be fairly asked, whether the condition of the blood may not be always produced by the inflammation. We are, however, quite of Rokitansky's opinion, that while, in many instances, there is no doubt that such is the case, yet that there are numerous others in which the local inflammation is the result of a foregoing crasis. The marked disproportion that is sometimes observed between the hyperæmia and the exudation, and the early occurrence of the latter in many cases, appear to us to argue strongly in favor of this view.

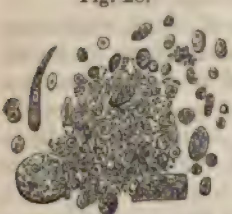
Rokitansky recognizes an aphthous variety of the fibrinous crasis, which gives rise to the exudations of muguet, diphtheritis, some dysenteries, and of hospital gangrene. These are manifestly outpourings of deteriorated diseased fibrin on various surfaces, rather than products of local inflammation. The alteration of the blood in these instances must certainly be primary.

THE TUBERCULOUS CRASIS—TUBERCLE.

The product of this crasis, from which it has its name, is the well-known substance, which, on account of its frequently spherical shape, is called tubercle. This we have not yet described; and though Rokitansky places it among the organized new formations, yet we think it will be more convenient to make mention of it here, in connection with our remarks upon the condition of the blood in which it originates.

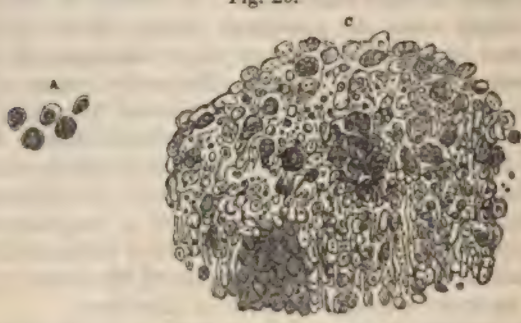
Tubercle, or tuberculous matter is, in almost all cases, an exudation of protein material, which speedily passes into the solid form, and never proceeds beyond the lowest grade of development. It very commonly assumes a spherical shape, which appears to depend partly upon its enlarging from its original magnitude by successive accretions to its surface, and partly on the nature of the tissue in which it is deposited. There are two principal varieties of tuberculous matter, distinguished as *gray* and *yellow* tubercle. The former, sometimes called gray granulations, are about the size of a millet-seed, roundish, resisting under pressure, of a grayish, semi-transparent aspect. The microscope shows them to consist of a basis-substance (blastematous), which is solid and homogeneous, and serves as the uniting medium of certain corpuscular elements. These are granules commonly of oily aspect, nuclei, oval, or more elongated, generally feebly formed, and cells, which are, for the most part, very few in number, and probably not to be regarded as any essential part of the tubercle itself. The globules of tubercle, which M. Lebert describes as characteristic of this morbid product, are nothing more than the ill-

Fig. 28.



Gray tubercle; miliary granulation.

Fig. 29.

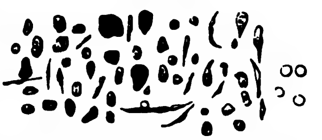


Yellow tubercle; crude mass.

developed nuclei just mentioned. Rokitansky applies to them the following epithets: "anomalously shaped, irregular, as if gnawed, angular, bent, constricted, rudimentary, stunted." The elements of the tissue in which it is deposited, are often found imbedded in the mass. This, however, scarcely applies, except to those which are not very readily

destroyed, as fibres. No vessels are ever found in separate tubercles; some traces of those belonging to the tissue may be imprisoned in the interspaces of several aggregated together. Yellow tubercle forms masses of varying size, but generally somewhat larger than those of the gray, equalling, perhaps, a hemp seed, or a pea, in magnitude. They are from the outset opaque, of a whitish-yellow color, of rather brittle consistence. Their microscopic structure is nearly identical with that of the preceding variety, only that they contain more diffused granular matter. Their relations, also, to the surrounding textures, are quite similar to those above mentioned. The yellow tubercle, which Rokitansky denominates the croupo-fibrinous, in opposition to the gray, which is the simple fibrinous, undergoes two metamorphoses of very great importance; one is that of *softening*, the other that of *cretification*. Softening consists in the texture of the mass becoming more lax and moist, with notable increase of size, the change proceeding till it breaks up into a yellowish, diffluent, cheesy mass, which finally becomes a thin, whey-like fluid, of acid reaction, containing minute flocculi. The change seems first to affect the homogeneous basis-substance, which dissolves into a kind of fluid, loaded with pulverulent molecules; the corpuscular elements in consequence of this are set free, and, at the same time, are themselves more or less corroded and dissolved. The softened tubercle thus consists of (1) a fluid loaded with diffused granulous matter; (2) traces of altered nuclei and cells; (3) free oil in the form of various-sized drops. It may also contain debris of the tissues. The cretifying change consists in the gradual deposition and liberation of calcareous particles in the tuberculous mass, together with simultaneous absorption of the animal matter, and consequent decrease in size. It is said by Rokitansky never to take place except in softening, or softened tubercle; but this is, probably, too absolute an assertion. The cretified tubercle very often remains as a hard, irregular mass, surrounded by indurated tissue, and appears to be insusceptible of further change; sometimes,

Fig. 30.



Isolated tubercle corpuscles. On the right are four blood-globules.

Fig. 31.

Fig. 32.

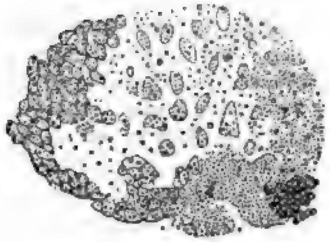


Fig. 33.

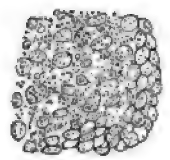


Fig. 31. Tubercle corpuscles from the peritoneum. *a*. The same, after the addition of acetic acid.

Fig. 32. Tubercle corpuscles, granules, and molecules, from a soft tubercular mass in the lung. 250 diameters linear.

Fig. 33. Tubercle corpuscles, from a mesenteric gland.

however, absorption proceeds further, and almost the whole of the deposit is removed. When this is the case, however, it is probable that

Fig. 34.

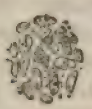


Fig. 35.

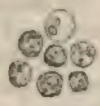


Fig. 36.

Fig. 37.

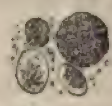


Fig. 38.

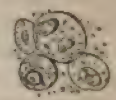


Fig. 39.

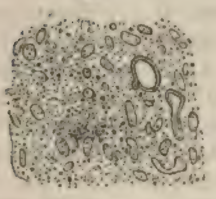


Fig. 40.

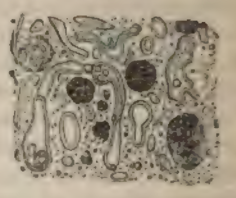
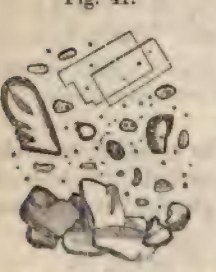


Fig. 34. Tubercle corpuscles from the lung.
Fig. 35. Pus-corpuscles. One shows the double granular nucleus after the addition of acetic acid.
Fig. 36. Plastic or pyoid corpuscles.
Fig. 37. Granular corpuscles from cerebral softening.
Fig. 38. Cancer-cells from the uterus. 250 diameters linear.
Fig. 39. Structure of the central portion of a tubercular mass, imbedded in the cerebellum.
Fig. 40. Structure of the external portion of the same mass, where it was in contact with softened cerebellar substance. 250 diameters linear.

absorption had predominated over the deposition of calcareous matter from the first. The only metamorphosis, according to Rokitansky, which the gray tubercle undergoes, is a kind of drying up into a hornlike substance, which, in some cases, is also the seat of calcareous deposit. This he calls *obsolescence.* It has been very commonly held, since the time of Laennec, that the gray tubercle, or gray granulation, was the nascent phase of the yellow: Dr. Walshe, after careful examination, maintains this view; Hasse and Rokitansky reject it. The latter regards the two as essentially distinct, though very frequently combined together in the same tubercle, in varying proportions; and remarks, with much reason, that it is an error to look upon these differences in composition as stages of transition, or conversion of one into the other. The apparent softening of the gray tubercle, when it occurs, is not dependent upon an alteration in its own substance, but in that of the yellow mingled with it. It is very interesting to remark how the behavior of the two kinds of tubercle corresponds with that of the fibrin, from which they seem to be derived. The gray resembles healthy fibrin in its tendency to contract and shrink up into an indurated mass; the yellow, like the croupous fibrin of coagula and exudations, tends to soften and break up into a fluid substance. Moreover, as the masses of croupous fibrin begin

Fig. 41.



Fragments of phosphate of lime, crystals of cholesterin, and tubercle corpuscles, from a cretaceous mass in the lungs.

to soften in their central part, so we find does the yellow tubercle. Dr. Carswell and others consider that inflammation and suppuration taking place in the tissues surrounding the tubercles, are the chief cause of its softening and breaking down. This Rokitansky denies; but though we believe with him that the softening change is one inherent in the tubercle-substance itself, yet we think the hyperæmic movement taking place around it must, at least, give an impulse to the process. As each tubercle, or group of tubercles, undergoes softening, the space which it occupied becomes the cavity of a minute abscess, the contents of which, sooner or later, are evacuated. The tissue involved in the tubercle is, of course, destroyed, together with it, but rather in the way of necrosis than of ulceration. The tendency of the tubercle to soften differs very greatly in different cases. Sometimes it is scarcely deposited before it begins to break down, sometimes it remains very long in its original (crude) state. The influence of the inflammation set up around tubercles upon their progress varies very much, chiefly according to the degree of the tuberculous dyscrasia. If this be very great, the result of the induced hyperæmia will be the infiltration of the bordering tissues with tuberculous matter of the lowest kind, tending to rapid diffluence, and involving in its destruction that of the infiltrated tissue. The increase of a tuberculous cavity in this way may be most rapid. On the other hand (and herein is contained a truth of the utmost interest to the practitioner), if the dyscrasic condition of the blood be slight originally, or if it have been amended by well-directed treatment, inflammation gives rise to the exudation of fibrin, which develops itself into the so-called induration tissue, or fibroid callus, which either surrounds and capsulates the tubercle, or forms a wall to and contracts the cavity, if one has formed. The surrounding tissues are often much puckered by the shrinking in of the fibrinous deposit. There occur occasionally, especially upon serous surfaces, small granulations which have much the aspect of tubercles, but which, in their progress, assume more of a fibroid texture; these may be regarded as specimens of an intermediate condition between tubercle and fibrinous exudation, and are, in this light, of great interest. Tubercle seems sometimes to be deposited in the way of infiltration; that is to say, it no longer forms the small characteristic *tubera*, from which its name is derived, but appears as a uniform mass which had been effused into the tissue in a fluid state, and had solidified there. The common tuberculization of the absorbent glands is very much of this kind; it is seen, however, most strikingly, in the lungs, a whole lobe or more of which may appear to be converted into a tuberculous mass. Sometimes this appearance depends on the part being occupied by numerous tubercles, crowded together; but then there can always be distinguished on a section interposed layers of pulmonary tissue which are not seen in real infiltration. The only doubt as to the real nature of apparent tuberculous infiltration arises from the great similarity between chronic pneumonic consolidation and this state, so that Dr. Walshe is inclined to consider them identical. Our own belief is, that in a person whose blood is in a high degree affected by the tuberculous dyscrasia, inflammatory hyperæmia may result in the exudation of a material which corresponds closely with tuberculous, but

is less inclined to soften and break down. The seat of tubercle in the vast majority of cases is on the exterior of the vessels, but in their immediate neighborhood; its blastema is a true exudation, but, inasmuch as it coagulates with great rapidity, it is not able to penetrate for any distance through the substance of a non-vascularized tissue. Hence, we

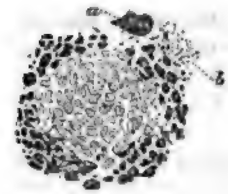
Fig. 42.

Section of a gray granulation in the lung after the addition of acetic acid, showing the pulmonary air-vesicles filled with tubercle corpuscles. 250 diameters linear.

do not find tubercle in cartilage. In very rare instances, coagula of tuberculous character have been seen *within* the vessels; but these, no doubt, underwent a morbid alteration after being formed; and there is not the least evidence to show that anything resembling tuberculous matter has even been detected in the blood. The microscope can discover nothing peculiar in the blood of phthisical patients, nor has chemistry detected any characteristic alteration in its protein compounds, which we might reasonably expect to find primarily affected. The exudation of tubercle-blastema may take place most gradually and imperceptibly, with scarce a trace of constitutional disturbance, or it may occur in a rapid, tumultuous manner, with all the symptoms of an acute illness; between these extremes the most various grades are observed. The more rapid the deposition of tubercle, the more is it associated with hyperæmia and inflammation. In fact, though the production of tubercle be quite independent of inflammation, and though inflammation, in the great majority of cases, is only secondary, and excited by it, as a cause of irritation, yet, when set up, it has a powerful effect in hurrying the exudation of tubercle, and that of such a kind as tends to rapid softening and decay. Commonly, the gray tubercle is the first to appear; sometimes, however, the yellow, in the miliary dispersed form; afterwards, as the dyscrasia increases, the exudation consists of yellow tubercle mingled with the gray; and, finally, of yellow tubercle alone. Tubercle may be deposited, we believe, in extravasations of blood, or, at least, its blastema may be mingled with blood; the changes which the latter undergoes, suggest to Rokitansky the

name of *pigmentary* tubercle, as distinguishing it from the more common varieties. Melanic matter, however, is often found, in small quantity, in the latter also. The following remarks are of much interest and importance relative to the co-existence of tuberculous disease with other affections.

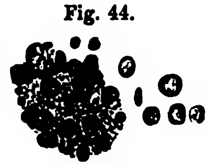
Fig. 43.



Corpuscles mixed with pigmentary matter, in a small tubercle taken from the peritoneum. *a.* Irregular masses of black matter, which may be broken down into (*b*) granular and molecular matter. 250 diameters linear.

Cystic growths are not often associated with tubercle, and the same is true of cancerous; when the latter are present together with tubercle, they are, in most cases, of secondary origin to it. Rokitansky contrasts the frequency of tuberculization of the lungs with the rarity of pulmonary cancer; the frequency of ovarian, gastric, and rectal cancer, with the rarity of tuberculous deposit in these parts. These and other facts indicate that the one morbid process tends to exclude the other. Typhus and the exanthemata, he states, do not commonly attack the tuberculous, but they are very apt to be followed by tuberculous disease. Sufferers from intermittent fever, goîtrous disease, and rachitis, seem to be, *pro tanto*, less liable to tuberculous affection. The non-coexistence of aneurismal and tuberculous disease depends, in Rokitansky's opinion, on the exhaustion of the fibrinous constituent of the blood, by the deposits taking place on the inner surface of the sac. An especial immunity against tubercle is afforded by an abnormally venous condition of the blood, from whatever cause this may come to pass. Congenital malformations of the heart or great bloodvessels; morbid alterations of the same; deformities of the chest, producing contraction of its cavity; annihilation of the function of one lung by pleuritic effusion; abdominal growths, preventing the free descent of the diaphragm; chronic pulmonary catarrh; emphysema and bronchial dilatation, have all been observed as exercising an unquestionable counter influence against the development of tubercle; and in all these conditions the free oxygenation of the blood is more or less interfered with. The undoubted effect of pregnancy in delaying the advance of tuberculous disease of the lungs, is explained by Rokitansky on the same principle of impeded, and consequently imperfect respiration, inducing a venous condition of blood; and he refers to the great production of fibrin, which takes place after parturition, as confirmatory of this view—tubercle being regarded as a fibriniform product. Respecting the identity of tuberculous and scrofulous matter, there can be no doubt. They have the same elementary composition, they undergo the same changes, they are produced in the same way, and produce the same effects on the tissues in which they are deposited. Generally, it may be said, that the deposit in the absorbent glands and bones passes for scrofulous; that in the lungs or brain, for tuberculous matter—both being essentially what we have described as yellow tubercle. The name seems to depend almost entirely upon the form. In

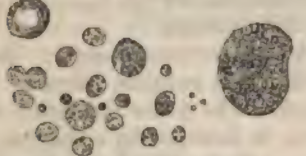
Fig. 44.



Scrofulous matter from subcutaneous deposit.

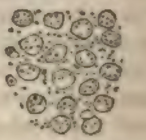
adults, tubercle is found in the various organs in about the following scale of frequency: Lungs, intestinal canal, lymphatic glands (especially the abdominal and bronchial), larynx, serous membranes, brain, spleen, kidneys, liver, bones, and periosteum, uterus and Fallopian tubes, testicles (with the prostate gland and vesiculæ seminales), spinal cord, voluntary muscles. In children, Rokitansky states, the lymphatic glands and spleen are most often affected, then the lungs, and after these the brain, &c. MM. Rilliet and Barthez assign, as in adults, the primary place to pulmonary tubercle. According to them, however, the lungs are not so invariably affected as M. Louis's well-known law declares them to be in adults; as, in forty-seven out of three hundred and twelve instances, they were exempt, while tuberculous deposit was found in other organs. It is to be observed that the above scale of frequency of the occurrence of tubercle in adults does not express correctly the

Fig. 45.



Scrofulous pus—a large glomerulus is shown, and some oil drops.

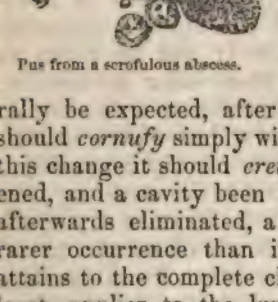
Fig. 46.



Scrofulous pus from a lymphatic gland. 250 diameters linear.

different tendency of the various organs to *primary* tuberculosis. Rokitansky places in this respect the lungs and lymphatic glands first, then the urinary organs, the bones, the testicles, &c.; while the intestines, the larynx, the spleen, and the liver, occupy the lower part of the scale. The question as to how far, and in what way tuberculous disease is curable, is of course of the greatest interest. As an exudation, it seems credible that tubercle should liquefy and undergo absorption; but it has been very generally doubted whether this ever actually occurs. Dr. Walshe, whose authority is great on this point, believes that absorption, under favorable circumstances, may take place, but acknowledges it to be a rare event. Probably the most favorable result that can generally be expected, after tubercle is once deposited, is either that it should *cornufy* simply without having undergone softening, or that after this change it should *cretify*. After tubercle in any quantity has softened, and a cavity been formed from which the tubercular detritus is afterwards eliminated, a cure may still take place; but it is a much rarer occurrence than in the two former cases, and perhaps never attains to the complete closure and cicatrization of the cavity. This, at least, applies to the lungs; in other parts, there is no doubt that a tuberculous ulcer may heal up and cicatrize. The production of tuber-

Fig. 47.



Pus from a scrofulous abscess.

cle sometimes takes place, as observed above, with very great rapidity, constituting what is termed *acute tuberculosis*. It is remarkable that the symptoms in this condition very closely resemble those of typhus fever (*v.* case in Dr. Walshe's work on Diseases of the Lungs and Heart, p. 409). The tubercle is of the gray miliary kind, is widely and uniformly scattered throughout the lungs, and is often deposited in other parts also.

As a sequel to the foregoing account of tubercle, we may describe here a somewhat analogous deposit, which is not very unfrequently found in the organs of those who are the subjects of general cachexia. It appears as a solid blastematous mass, infiltrated among the tissues of a part; semi-transparent, or verging on a whitish opacity—presenting, under the microscope, an amorphous, flaky basis-substance, together with scanty nuclei. It is commonly deposited in a part in considerable quantity, and gives rise to the appearance of hypertrophy, though at the same time the natural elements of the tissue are compressed and atrophied, often to a great extent. An organ thus affected is bloodless, breaks with a sharp fracture, and strongly resembles bacon in appearance, from whence the term "bacony" is applied to the deposit by German writers.

The formation of this matter is not peculiar to the scrofulous diathesis, but it is observed in those who have become cachectic from any cause; as from the abuse of mercury, inveterate syphilis, habitual intermittents, or any severe drain upon the system. Rokitansky calls the deposit "albuminous raw blastema," and believes it to proceed from an undue quantity of albumen being present in the blood.

With respect to the real nature of the tuberculous crasis, we have scarce any exact knowledge. It is evidently a special dyscrasia, intimately connected, as we know, with causes of debility, and leading to the effusion of a matter, which shows only the feeblest traces of organization. This matter in many respects comports itself very differently to fibrin; so much so, that the one might almost be regarded as the antithesis of the other—supplanting it in the process of effusion, or itself replaced by it. Rokitansky, however, shows some weighty reasons for regarding tubercle as a modification of fibrin; and after a most interesting discussion, to which we would particularly refer (*v.* p. 522, German edition), concludes that "the arterial character—arterial elaboration of the fibrin—constitutes, above all, the cardinal feature of the tuberculous crasis." He also points out how, in consequence of the alteration of the nature of the fibrin, tubercle is continually deposited, even when the blood is very deficient in that constituent. All the fibrin that is formed is soon affected by the peculiar dyscrasia, and is thrown out in the form of tubercle. The rapid coagulation of tubercle-blastema, which must be effused in a fluid form, its tendency, when coagulated, to soften—its formation being favored by active arterialization, and prevented by a venous condition of the blood—are circumstances which indicate a real affinity between tubercle and fibrin. When we further reflect that various debilitating causes are found to increase the quantity of fibrin, and also that the same are potent in causing the production of tubercle,

we gain further evidence to the same effect. No doubt, however, even before that peculiar modification of the fibrin has occurred, which leads to its excretion in the form of tubercle, a special impress is, at least in many cases, stamped upon the system, which betrays to the instructed eye the future evil. The tendency and proclivity to disease is there, it may be, long before its actual development. This unexplained proclivity it is which constitutes the scrofulous diathesis.

A condition of the blood, characterized by deficiency in fibrin, excess of albumen, and for the most part also of blood-globules, is termed by Rokitansky venosity, or albuminosis. Simon designates it hypinosis, in contrast to hyperinosis, which implies an excess of fibrin. Rokitansky describes under this head several crases, in which the blood partakes of the hypinotic character; but we shall do no more than enumerate them, as we think he ascribes far too much to the apparent qualities of the blood, and does not take sufficient count of the unseen but essential derangements: "Hypinotic blood is in general a thick, sticky, dark red fluid; contains no coagula, or only small, soft, sticky, gelatinous ones, which include much cruor." It is apt, under peculiar circumstances, to undergo various changes, such as septic destruction of the albumen, in which case necræmia takes place; or a croupo-fibrinous or pyæmic condition may supervene; or a tendency to the effusion of acid fluids and acute softening of tissues. Dark hypostatic stains, speedy putrefaction, transitory rigor mortis, a lax state of the solids, are observed in the bodies of those who die with this condition of blood.

The subordinate hypinotic cases are—(1) plethora; (2) the typhous; (3) the exanthematic; (4) that existing in certain diseases of the nervous system; (5) drunkard's dyscrasia; (6) cancerous dyscrasia.

We may mention, with regard to the drunkard's dyscrasia, that, when chronic, it presents a remarkable dark color, and inspissation of the blood, with excessive quantity of oil. Fat is formed abundantly in the subcutaneous tissue, and in other parts. The liver, the muscles, and even the bones are either encroached on by the fat, or undergo some degree of fatty degeneration. The cerebral membranes are apt to become thickened, the brain itself to be atrophied. Chronic fluxes, from the mucous membranes, especially the bronchial and intestinal, are very common. The crasis often undergoes change to the croupo-fibrinous.

We would recommend the doctrine of crases of the blood to the careful thought of our readers. No doubt it may easily be carried too far; but we think cases will often present themselves, in which an apparent inflammation and manifest exudation will be better explained and managed by the ideas which this doctrine suggests, than by the most vigorous anti-phlogistic proceeding.

CHAPTER III.

TEXTURAL CHANGES.

We now come to consider certain changes, to which most of the various organs of the body are liable, in a general way. These changes are essentially textural, and result from various disturbances of the normal degree and kind of nutrition. They are also for the most part slow and gradual in their course, and are thus termed chronic. They are intimately dependent on the condition of the blood, so that their consideration follows very properly on that of the diseases of this fluid.

Hypertrophy, as its etymology signifies, conveys the idea of increased nutrition and growth in the part affected. The term, however, is sometimes applied to parts which are simply enlarged, and it is essential to observe that this enlargement by no means necessarily constitutes hypertrophy, but may, instead, be attended with the opposite condition. This makes it necessary to distinguish *real* from *apparent* hypertrophy. In the former, the characteristic tissue of the part is enlarged, and more developed; if it be a muscle, the muscular fibres grow larger, and attain to greater energy of contraction, if it be a kidney, more renal tubes are formed with corresponding bloodvessels. The size of the organ is not only increased, but its working power too; the muscle can raise a greater weight, and the kidney can produce more secretion. But if a liver or spleen be enlarged by ever so great a quantity of the peculiar matter termed "bacony," which is deposited interstitially between the elements of the tissue, their functional power is only thereby lessened, and deteriorated, for the simple reason that the new substance has pressed upon and caused wasting of the natural structure. It is, therefore, necessary in every case to ascertain what is the nature of the enlargement of a part before we pronounce it to be truly hypertrophied. Mere distension of a hollow organ, of course, is not hypertrophy; a huge emphysematous lung, or hydrocephalic brain are not really enlarged, but rather diminished in actual capacity. Great congestion of a part with blood may give it the appearance of being hypertrophied, but this again is only another kind of distension. Real hypertrophy requires a free supply of healthy blood, and is commonly attended with enlargement of the vessels of the part; this is not the case in apparent. The cause of real hypertrophy seems to be always the increased exertion of the organ, more than usual effort is demanded of it, and, according to the law of the circulation which we have noticed, more blood flows to the part than usual; this, if the organ be in a healthy state, not only supplies its waste, but furnishes material for increase and development. The heart

in various diseased states of its valves, the urinary bladder in stricture of the urethra, the remaining kidney when one has been destroyed, the muscles, and even the solid bones themselves, when long and actively exercised, afford excellent examples of true hypertrophy.

This process, though in several instances it brings about an abnormal state of the part, is yet for the most part not to be considered in the light of a disease. It is really a compensatory effort made by the system, to obviate as far as possible the evils that arise from some damage that an important part has sustained. Thus, if we find the walls of the heart greatly thickened, and its power proportionately increased, we should naturally fear that such an abnormal increase of power would prove a cause of danger to the system, and would probably induce hemorrhage in the brain, or elsewhere; but if we know that at the same time there exists regurgitant disease of the mitral or of the aortic valves, then, we see, that the hypertrophy, so far from being attended with danger, is useful and necessary to enable the circulation to be carried on against such impediments.

Atrophy is the opposite condition to hypertrophy; and is commonly conceived of as implying a wasting and diminution of the part. Atrophy, however, may have taken place to a great extent, without any diminution, but an increase of size. These are the instances of false hypertrophy, to which we have above alluded. In a few instances atrophy is a natural process, as in the disappearance of the thymus gland when the age of early infancy is passed. Inactivity of a part, obstruction of its bloodvessels, failure of its own vital energy, continued pressure upon its surface, are all recognized causes of atrophy. A muscle, if unused, becomes small and pale, and its tissue degenerates; the bones of a paralytic limb lose in density and strength, and in compactness of tissue; the brain in second childhood shrinks within its bony case, and leaves a space occupied by serum. Obstruction of the arterial branch leading to a part of the kidney will cause wasting of the epithelium of the tube in that part, ligature of the thyroideal arteries has caused considerable diminution of a goîtrous tumor. Thinning of the walls of the heart, renal degeneration, the fall of the hair, and the general decay of advanced age, are instances of atrophy from failing vital energy of the tissues. With the effect of pressure in producing atrophy, all are familiar; it is well exemplified in the absorption that takes place from the pressure of aneurism, which affects not only the soft parts, but the bones themselves. In most cases, atrophy is an actually morbid process, and is attended by a change in the condition of the elementary parts of the tissue which attests the unhealthy character of their nutrition. Atrophy often occurs as a secondary process, induced by some primary one, which may have been attended with apparent hypertrophy. Of this, the liver in the earlier and later stages of cirrhosis furnishes an instance.

Induration and *softening*, are terms that are commonly employed to express changes that have occurred in the consistence of various organs, rendering them more or less firm and dense than natural. They are, of course, very general in their meaning, and of themselves tell nothing as to the pathological condition of the part affected. This must depend

entirely on the causes of the changes in question. The commonest cause of induration is the effusion of fibrinous material into the interstices of a tissue; if this does not liquefy and become absorbed, it passes into the state of fibroid texture, and being blended with the elements of the part, it occasions a more or less considerable increase of density and toughness. Instances of this are extremely frequent in the lungs around tuberculous deposits, in the cirrhotic liver, and in the areolar tissue around ulcers. The tissues involved in the induration matter, as it is often called, are very apt to become atrophied, partly in consequence of their supply of blood being cut off by obliteration of the vessels distributed to them, partly from the effect of atrophic pressure itself. According to its seat, induration may be of trifling consequence, or very serious; in the general areolar tissue of the body, it may only cause slight impediment to the free movements of a part; in the valves of the heart it is a common cause of secondary disease, dropsy, and death. Textures are often rendered harder and firmer by other deposits than simple fibrinous, as by tuberculous, bacony, calcareous, but to these the term induration is not so strictly applicable.

Softening of a part may be brought about by very various causes. It is almost an invariable effect of acute inflammation actually existing; it is also found as the result of inflammation that has in great measure subsided; it occurs from deprivation of blood, as a kind of atrophy, and probably, in some cases, as a local result of a general cachexia. In all cases it involves a considerable deviation from the state of healthy nutrition, and if it proceeds far, may easily occasion a breaking down and destruction of the tissue. The distinction of various kinds of softening, especially of the red, or inflammatory, has often been considered difficult, but may generally be made with certainty by means of microscopical examination, which discovers in the former decided traces of exudation. Softening is connected with the hypinotic condition of the blood, and its subordinate crases, especially the typhous; induration, on the other hand, with the fibrinous crasis. Softening is more prone to occasion speedy destruction of textures and fatal disorder. Induration, to produce gradual changes whose effects are slowly and gradually manifested. Softening appears as a process of decay, and affects not only natural structures, but new formations, and even as we have seen tuberculous deposits and fibrinous coagula. Induration, on the contrary, though involving some degree of atrophy, tends to preserve the parts which it affects from entire dissolution.

Degenerations are changes of an essentially chronic nature, latent in their origin, and obscure in their progress, until they have produced such deteriorations of structure as give rise to prominent secondary phenomena. Those with which we are most acquainted are the fatty, fibrous, and calcareous. They are of extremely frequent occurrence, but their nature has scarcely been recognized until of late.

Fatty degeneration consists in the replacement of the healthy tissue of a part, by drops, or molecules of oily nature, which are deposited, as it seems, instead of the natural material. This character distinguishes it from fatty accumulation, which may take place to a great extent in the interstices of a tissue so as to overlay and conceal its ele-

ments. In true fatty degeneration there is always destruction of tissue, which does not occur when there is merely an increase of oil in the substance of the part. A muscular fibre thus affected shows the sarcous elements, the real contractile tissue within the sarcolemma, replaced by glistening oil-particles, so that the functional power of the organ is *pro tanto* destroyed. The hepatic cells in true fatty degeneration not only fill themselves with oil, but fuse together with others, and break up into granulous films, entangling oil-drops; this destruction does not occur when they simply become loaded with oil from the presence of a large quantity of this substance in the food. The process by which fibrinous coagula, or extra-vascular deposits are broken down and dissolved, seems to be in some measure of the nature of fatty degeneration; there is commonly much free oily matter visible in the softened mass, and the exudation corpuscles seem to be thoroughly charged with it. Fatty degeneration is clearly a kind of atrophy, but not identical with the simple form; we have seen muscular fibres of the heart which were simply atrophied, and had lost their transverse striation entirely, which yet did not contain a single particle of oil. The prevalent opinion respecting the nature of fatty degeneration is, that there occurs a true conversion of the albuminous substance of the tissue into fatty matter, just as when adipocire is formed out of flesh immersed in water. We are rather inclined to believe that the change is effected in the way of an unhealthy nutrition, oil being deposited in the blood in the place of nitrogenized matter. However this may be, it is important to distinguish the following conditions in which the quantity of fatty matter in and upon a part is greatly increased. (1.) Mere accumulation of adipose substance in and around an organ, the tissue remaining healthy, (2.) Accumulation of adipose tissue in the same way, but with atrophy of the proper structure. (3.) Increase of oil in the elementary structure of a part without atrophy, or breaking up. (4.) True fatty degeneration, in which the structure is more or less destroyed, and its elementary parts converted into oily matter.

Fibrous Degeneration is somewhat allied to Induration, and is probably connected with the existence of a fibrinous crasis. It occasions the gradual thickening of serous membranes and of areolar tissue by the formation of an imperfect kind of fibrous structure. This may attain a considerable thickness, and then by its dead white aspect resemble very much a layer of cartilage. The capsule of the spleen is sometimes thus altered, and has been wrongly said to have undergone cartilaginification, for there is no real similarity between this substance and cartilage. The white patches formed on the surface of the pericardium and in the capsule of the liver, are produced in this manner, and so is also that thickening of the Glissonian sheaths, which give rise, in many cases, to cirrhosis. The fibres are probably formed, in part, directly out of the effused blastema, in part, also, by nuclei, developing short fibres, which unite, as Henle has described. This latter mode of formation is often observed in the spleen. The chief difference between induration and fibrous degeneration consists in this, that in the former, a notable quantity of blastema is effused, which becomes the induration matter, and compresses and atrophies the adjacent texture; in the latter, there seems to be scarce

any perceptible exudation, as it takes place slowly, and passes at once into the condition of fibre. Induration may affect any tissue, while fibrous degeneration is chiefly seen in membranes. Cartilage, however, is liable to a fibrous transformation of a somewhat different kind, which will be hereafter noticed.

The *Calcareous degeneration* rarely occurs as a primary change, it is almost always secondary to some other. Especially, it seems to be consequent upon a fatty degeneration of the arteries, to which the term *atheroma* is applied, and which may occur at any period of life, while the calcareous change is seldom observed very extensively except in advanced age. We have already alluded to the calcifying process, under the head of tubercle, as one of the metamorphoses which that deposit might undergo, and we shall find hereafter that it affects other formations also. It is often spoken of as ossification, and, indeed, not altogether without reason, as the "lacunæ" characteristic of bone are found in this substance also; they are, however, irregularly and imperfectly developed. The earthy matters deposited are principally phosphate of lime and magnesia, and carbonate of lime; Rokitansky considers that they are not so much new deposits as precipitations from their natural combinations with animal matters. Calcareous deposition seems generally to take place in parts whose vitality has been considerably lowered by previous morbid processes within them. Thus it is common, in lymphatic glands which have been the seat of scrofulous disease, in obsolete croupo-fibrinous deposits, in the coats of arteries which have begun to be affected by atheroma, and in the valves of the heart under similar circumstances. The atheromatous condition, which we shall describe more particularly when we speak of the diseases of arteries, may either terminate in softening and breaking down of the arterial coats, or in calcareous deposition; both of these changes often coexist, but the latter predominates in old age. We think, however, that deposition of earthy matter may take place to a great extent, so as to produce the ossification so common in the vessels of the aged, without previous atheromatous or fatty degeneration. The quantity of earthy matter in the bones becomes greatly increased in later life; it is even deposited in the so-named permanent cartilages, and it is, therefore, not surprising that it should also affect the walls of the vessels. This degeneration probably is occasioned solely by a failure of assimilative nutritive power in the tissue itself.

CHAPTER IV.

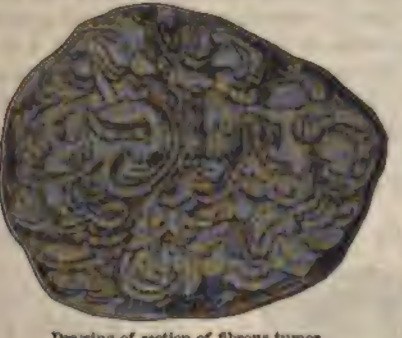
NEW FORMATIONS.

It is difficult to give a perfectly exact definition of the class of new formations; for we shall exclude from it many productions which are not found in the healthy organism, and shall include in it some which are but the result of the action of parts normally existing. Thus, we shall not mention the excessive production of fat-cells, which takes place in general obesity, as an instance of new formations, while we shall consider as such the distension of a sebaceous follicle into an encysted tumor. This defect, however, is common to all arrangements. Nature presents us readily with distinct types of different classes; but rarely, if ever, does she define and separate her groups by any exact limitation, and the rigid taxonomist wearies himself in the search for that which does not exist. The idea which is conveyed in the term "tumors," seems, in a general way, most descriptive of the class now before us, which may be said to include all new *prominent* or otherwise *apparent local growths*. The character of growing excludes tuberculous and other deposits, and concretions. We shall follow, in the main, the arrangement of the various kinds of new formations which Rokitansky has adopted, endeavoring to set forth their distinctive features as far as possible, and yet recognizing the frequent insufficiency of any structural or chemical peculiarities that we can observe—to explain, or even diagnose, the essentially different natures of different specimens we may meet with.

(1.) *Fibrous Tumors.*—These constitute a group with tolerably well-marked structural characters, but shading, almost imperceptibly, into other species of very different nature. They are essentially made up of fibres, more or less closely resembling those of areolar tissue, but appearing, in very various stages of development, in different specimens. Sometimes the fibres are tolerably distinct and separate; more often so interlaced and blended together, or so imperfectly evolved, that they cannot be made out as such. Sometimes the nuclei, with which the structure is loaded, seem to be simply imbedded in a granulo-homogeneous blastema, the whole forming a dense solid mass; sometimes the blastema is divided into fibres, very similar to those of organic muscle; and sometimes, again, but more rarely, the blastema is broken up by fibrillation into bundles of filaments, identical with those of white fibrous tissue. Yellow elastic fibres are not unfrequently mingled with the white, and seem to be developed from the elongated nuclei. Much difference is observed in the chemical behavior of tumors of this class. Those which consist of fully developed fibres yield gelatin, while from those

which consist of muscle-like fibre, or of an undivided blastema, none can be obtained.[1]

Fig. 48.



Drawing of section of fibrous tumor.

Fibrous tumors differ much in their degree of connection with surrounding parts; sometimes they are quite blended with them by continuity of tissue; at others, they are easily enucleated. They have

Fig. 49.

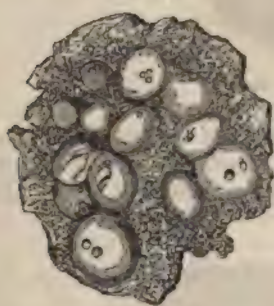
Fibro-fatty tumor. The upper figure shows fat-cells imbedded in fibrous tissue; the left lower one represents a fat-cell capsulated by fibres; and the right some separate fibres.

very few vessels indeed; so few, that it is matter of surprise how some large masses maintain their vitality. These tumors develop themselves in very different parts of the body, usually in such as normally contain

[1] This is Vogel's statement; but we have certainly obtained abundance of gelatin from tumors consisting of undivided blastema.

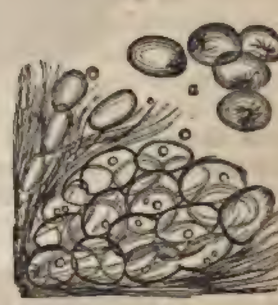
much fibrous tissue. The uterus is one of their most common *hábitats*, which probably depends on the similarity between the undeveloped muscular fibre of the organ and their own structure, so that a slight alteration in the conditions of nutrition might cause the common blastema to take the form of fibrous tumor, rather than of uterine fibre. Several fibrous tumors may exist in the same organ; but it is rare that they coexist in separate organs. They are not liable commonly to any great degree of change. Inflammation may occur, characterized by injection and softening of the part, and probably by the presence of exudation corpuscles in it. Cretification is not unfrequent, and may either commence indifferently at any part, proceeding until the whole is converted

Fig. 50.



Fat-cells and granular matter, from a steatomatous tumor of the ovary.—Bennett.

Fig. 51.

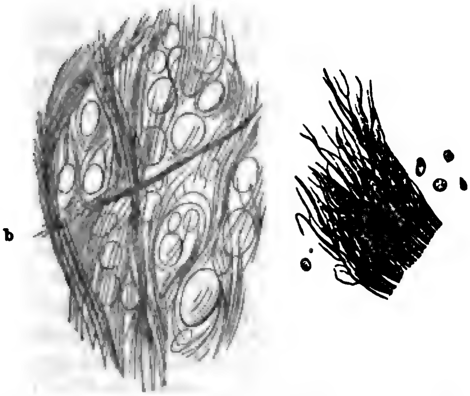


Structure of a fatty tumor removed from the back. Lipome. *a*. Isolated cells showing the crystalline nucleus of margaric acid.—Bennett.

into a calcareous mass; or it may be in great measure limited to the peripheral stratum, which it thus converts into a kind of shell, inclosing the rest. The calcareous degeneration affects tumors of very different size, and does not bear any relation to the age of the growth. Melanic matter is sometimes deposited abundantly in fibrous tumors. Cyst-like cavities, filled with clear fluid, are occasionally found in fibrous tumors, constituting thus a fibro-cystic variety. Another results from their combination with adipose tissue, of which we have figured a specimen. The form which fibrous tumors assume is mostly the spherical, with a more or less nodulated exterior. If, however, they grow near a free surface, they are very prone to become pedunculated. It is probably from this tendency, together with subsequent wasting of the peduncle, that fibrous tumors are occasionally found free in the cavity of the uterus. In a case which came under our notice, there was no trace of peduncle. The tumor, of large size, had greatly distended the uterine cavity, and had very slight connection with the parietes. The size which fibrous tumors sometimes attain is very considerable, some have weighed as much as thirty-five or thirty-nine pounds. Between proper fibrous tumors and instances of mere hypertrophy of the integument, there exist, or may occur, every intermediate variety. In proportion as the texture of the formation becomes dense, it is more abundantly permeated by vessels, so that some of these growths are highly vascular. Of this

kind is the rather rare *Keloid* tumor, a specimen of which we have lately had an opportunity to examine. It was situate on the back, was well defined, of a red color before removal, but pale after. It had much the

Fig. 52.



Fibro-cystic tumor from back: (b) after addition of acetic acid.

appearance of a thick red cicatrix, as if formed by a kind of transformation of the skin itself. The structure, under the microscope, appeared as a dense woof of fibrous tissue, resulting from a pretty thoroughly fibrillated blastema. Acetic acid brought into view numerous nuclei, all more or less elongated, some quite passing into streaks, as if about to form nucleus fibres. A good deal of free, oily matter was diffused through the mass. Another specimen we have lately examined consisted of a fibrillating but undivided blastema, imbedding numerous nuclei, for the most part elongated and streaky. It involved the corium of the skin, but the epidermis passed evenly over it. It was very remarkable that the deeper layers of the epidermis consisted of vertically elongated cells and nuclei, while the upper layers were of the usually flattened shape. This probably depended on the abnormal fibrefying tendency having affected the cell growth on the free surface of the basement membrane. Tegumentary tumors, as they are termed by Mr. Simon, seem to be properly included in the class we are considering, as they consist essentially of an increased growth of fibrous structure.

We may here allude to a group of tumors, for which Mr. Paget proposes the name of Recurring Fibroid. They are almost identical with common fibrous tumors, both in their naked-eye aspect, and in their microscopic characters, but show a remarkable tendency to return after removal. It is an extremely important and interesting circumstance, that the later produced tumors approximate much more in appearance and in behavior to the malignant character, than the original one. In one of Mr. Paget's cases, the last production was hardly to be distinguished by the naked eye from encephaloid (and, indeed, we doubt if it was different), though it still consisted of the same elongated fibre-cells. The chief pathological interest of these tumors consists

in the circumstance that they form a kind of transition between the so-called innocent and the malignant formations. According to our belief, the circumstance of structural resemblance to fibrous tumors, is no hinderance whatever to the possession of malignancy. A caudate cell may have this fatal gift as well as any other kind.

Epidermic and *epithelial* tumors constitute a well-marked class of new formations, which are of very frequent occurrence, and much practical interest. Warts and callosities of the skin are minor instances of this group, and consist simply in thickening of the epidermis, produced by accumulated layers of its scales. As an increased flow of blood to the part must take place, it is not surprising that the papillæ of the corium beneath should, in some of the more advanced cases, become hypertrophied and elongated, so as to project upwards into the little tumor. In condylomata, mucous tubercles, and similar vegetations, which are apt to form about the orifices of mucous canals, under the irritation of syphilitic and other discharges, the surface is commonly observed to be lobulated or papillar, the interior marked by a vertical striation, while some vascular ramifications extend up into each papilla. The structure of these is beautifully figured by M. Lebert. The surface of each papilla, as shown in his plates, is formed by a layer of closely imbricated epithelial scales; while the deeper parts consist of either less flattened cells, or, according to our own observation, of nuclei, lying close together in a granulous and amorphous blastema. This interior nucleated granulous tissue, we believe, is continued downward to the base of the growth, and encroaches on the corium of the skin; for we have never been able to observe any clear demarcation between the vascular loops and the surrounding cell formation. Almost the very same description applies to those tumors which are most common on the lips, and whose cancerous nature one has often too much reason

Fig. 53.

(A) Papillary prominence of epithelial growth.
(B) Epithelial tumour from lip.
From Lebert.

to suspect. These attain a much larger size, and are more manifestly vascular than the preceding, and their papillæ are more branched and grouped together, so that the surface resembles somewhat that of a

cauliflower. We believe it is not always possible to say, from the structural characters of these growths, whether or not they are malignant, or whether, if removed, they will return again. To this point we shall advert again, under the head of "Cancer." Horns are epidermic productions, which are occasionally formed upon the head, the forehead, or some other part of the body. They originate in the sebaceous follicles, whose epithelium, thrown off in unnatural and excessive quantities, and mixed with the fatty secretion, forms a conical mass, which protrudes from the orifice in the skin, and is pushed onwards continually by fresh accretions to its base. M. Lebert quotes a case in which the horn was six to seven inches broad at its base, and six inches long. A contusion, or ulceration of the skin, preceded the appearance of the tumor.

Melanotic tumors are not unfrequently spoken of by various authors, and occupy in their arrangements a place with other classes; but there is very great reason to doubt whether, properly speaking, any such thing ever exists. This is indeed generally admitted, and we now proceed to show why it is so. *Melanosis* (meaning thereby the deposit of black pigment) is an extremely common occurrence, and may take place in healthy tissues, in those which are variously diseased, and in new formations of any kind. The pigment is in the form of minute granules, or sometimes of almost dust-like molecules. It very commonly occurs free, though the particles may be more or less closely massed together, but is very often also contained in the interior of cells. There is nothing at all peculiar in these pigment-containing cells. They seem to be simply the natural cells of the organ, or of the new growth in which the deposit has occurred. Pigment granules are seen in the same part, both free and contained in cells, so that it is clear that their presence in the cells is of no special import.

Fig. 54.

Melanic deposit in cells of an engorged lung. Some of the cells contain oil-drops.

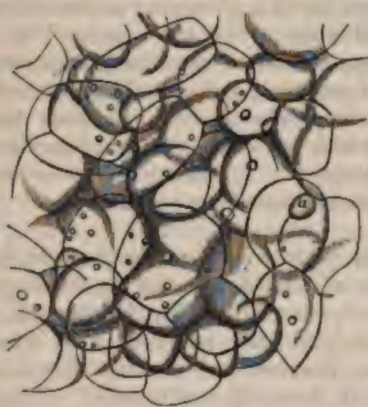
Rokitansky gives the following enumeration of localities, in which the melanotic deposit takes place: *In the lungs*, both in the air-cells (often in their epithelial particles), and in the connecting areolar tissue. When contained in the air-cavities, it is in all probability chiefly inhaled as carbonaceous matter, floating in the air. We examined some time ago the lungs of a man who had been a worker in a gunpowder manufactory, and found them to contain a very remarkable quantity of free black matter. In the *bronchial* glands, its quantity in them being usually in proportion to that in the lungs, it is rarely contained in cells. In the *mucous membrane* of the stomach and intestines, of the *uterus*, and occasionally of the *air-passages*, it is generally the result of the irritation of chronic catarrh, and is derived from altered hæmatin. In the *mesenteric glands*, coexisting with such deposits in the mucous tissue, it produces a slaty gray, or still darker discoloration, spotted or uniformly diffused. In the *sympathetic ganglia*. In the *skin*, either naturally, in the dark races, or as the local discolorations, termed "melasma." In new formations, as intravascular coagula, atheromatous patches, and their cicatrices, in hemorrhagic masses, false membranes of inflammatory

origin, tubercle, especially the hemorrhagic variety, colloid matter, and cancerous growths. Lastly, melanotic matter occurs in fluid exudations, and in the cavities of cysts. Rokitansky is of opinion, that the derivation of the black pigment from the coloring matter of the blood is a settled point. We are rather inclined to agree with him than with Dr. Walshe, who thinks "that the relation of true melanic cell-pigment to the constituents of the blood, is altogether unknown." It is quite certain that, in all the instances above mentioned, except that of the air-cavities of the lungs, the melanic matter proceeds in some way from the blood; but the question is, *how?* Is there first extravasation of the blood, exudation of its hæmatin, and conversion of this into the pigment; or is this produced from the liquor sanguinis, in some unknown way of secretion? We are inclined to think the latter is not unfrequently the case, and for the following reasons: (1.) It seems quite impossible to believe that the very considerable quantity of black matter, often found in the interlobular tissue of the lungs of old persons, should have proceeded from local congestions and extravasations, especially when there is no trace of previous inflammatory action. (2.) We are well acquainted with the changes which hæmatin does undergo in the splenic tissue and in the renal tubules, when blood has escaped out of the vessels; and we have far most commonly observed the color of the granules to be an orange or reddish yellow. The same is the case in the remarkable production of yellow matter, which takes place in the congested centres of the hepatic lobules. (3.) We have observed, particularly in the embryo of the fish, the development of pigment, and seen it commence by the appearance of a minute *free* particle of intense blackness, smaller than a nucleus, close by the side of a vessel; so also, in the abundant formation of black pigment which takes place in the liver of the frog at certain times, there is not the least reason for regarding it as specially derived from the red coloring matter of the globules, but much more for supposing it to proceed from some of the highly carbonized matters contained in the liquor sanguinis. In this instance, as in many others, truth lies intermediate between opposite opinions. The chemical composition of melanotic matter is not accurately ascertained, and probably hardly can be, owing to the difficulty of obtaining it at all pure, and perhaps also from variations in the composition of different specimens. It is clearly, however, a very highly carbonaceous substance—indeed, that obtained from the lungs of aged persons, by M. Guillot, seems to have been actually carbon. Dr. Walshe mentions that a specimen of softened melanotic tumor, which he examined, was not deprived of its color by acids or alkalies, only by chlorine. Strong nitric acid, however, with the aid of heat, turned it yellow. From the foregoing account of melanosis, it will readily appear that, from the very commonness of its occurrence, it is impossible to regard it as giving a really distinctive character to a tumor. The growth is essentially something else—a cancerous, or sarcomatous, or some other formation, and the melanotic tinging is accidental.

Fatty tumors, or Lipomata, as they are sometimes termed, are of frequent occurrence. They consist of normal fat-cells, closely packed together, and invested by a rather sparing quantity of common areolar

tissue. Occasionally, this investment is more developed, and constitutes a kind of enveloping cyst; occasionally, also, it dips down, and forms a cystoid covering to separate portions of the tumor. They occur most

Fig. 55.



Adipose tissue from a fatty tumor.

often singly, but not unfrequently several exist together in the subcutaneous tissue. They attain, occasionally, an enormous size, so that records speak of specimens several feet in diameter, and weighing 20—40 pounds. Their most common seat is the subcutaneous areolar tissue, especially in regions where fat is apt to collect, as on the buttocks, the thighs, the back and neck, &c. They have, however, been seen in many other parts, as beneath the scalp, in the submucous tissue of the stomach, intestines, bronchi, and in the underlying areolar tissue of the various serous and synovial membranes. In the knee-joint, especially, fatty growths have been distinguished by Muller as "lipomata arborescentia," in consequence of their branching form; this seems to result from their originating in the areolar tissue, and growing inward towards the synovial cavity. Lipomata have further been observed in the lungs, liver, and kidneys, and in the bones. The surface of lipomata is commonly lobulated—their form, for the most part, globular; they have a peculiar doughy feel, with some degree of elasticity. They grow slowly, and occasion inconveniences only by the pressure they exert on surrounding parts; when at last this distension becomes excessive, the skin covering the tumor attenuates, and ulcerates, and a sloughing sore may be thus produced, which may destroy life by exhaustion. It sometimes happens that a fatty tumor seated in the submucous tissue pushes as it grows the yielding membrane before it, and thus, acquiring a pedicle, hangs into the intestinal cavity. A *steatoma*, according to Rokitansky, is a fatty tumor, with a preponderating excess of areolar tissue, and hence of firmer consistence. Lebert applies the name to collected masses of fatty matter, not consisting of fat-cells, but of concrete fatty granules. Dr. Walshe says, that the steatoma is close in grain, inelastic, opaque, suet or putty-

like, composed of granular, amorphous, and non-vesicular fat. To this kind of tumor we think the name most suited.

Another variety of fatty tumor is the *cholesteatoma*, in which a substance allied to cholesterin is secreted in the interior of a fibroid cyst, lined internally by a delicate epithelium, which is probably the secreting organ. The contents are of a glistening mother-of-pearl aspect, though sometimes of a dull white; they are arranged in delicate concentric laminæ. The laminæ consist of superimposed strata of cells, rendered polygonal by mutual pressure, and resembling, except in size (being one-half smaller), the cells of sheep's-fat. Between the laminæ there are numerous crystals, which are of tabular and lamellar shape, and seem to consist of pure cholesterin. A similar matter is occasionally found on the free surface of cancerous and other ulcers.

Before we proceed to describe *vascular tumors*, which constitute our next class, we shall give some account of the development of new vessels in inflammatory exudations, and other blastemata. Two opinions are held at present as to the mode in which their formation takes place. By some, as Paget, Travers, and Simon, they are considered to be formed by "outgrowth from adjacent vessels." Small dilatations appear on the side of a vessel, increase in length, and at last meet and coalesce with similar diverticula in their vicinity; in this way a new capillary loop is formed, and the process is carried on in the same way. On the other hand, Rokitansky, Vogel, and Dr. Walshe agree in regarding the new vessels as originating spontaneously in the exuded blastema. We strongly incline to the belief that their view is the correct one, or at least that bloodvessels are formed in the latter as well as in the former manner. The following description is taken from Rokitansky and Vogel: In the substance of the exuded material there appears to the naked eye small roundish spots full of blood, from which there proceed in all directions minute streamlets filled with the same red contents. These primary channels are at first mere excavations in the blastema, and have no lining membrane; after a time they present the characteristic homogeneous coat of true vessels, and at a later period the external tunics are added. The blood-globules originating within their channels are more irregular in size, less exactly shaped, and have not the deep red color of the original ones. It seems certain that the primary blood-containing spaces and their offsets are not ramifying cells, for they appear in the blastema before the formation of cells, or even of fibroid tissue; the development of blood at certain points seems to be the only determining cause of their formation, just as it is in the embryo. Our knowledge respecting the influence which the nature of the blastema has on the production of new vessels, amounts to this—(1), that in blastemata undergoing similar developmental changes, the tendency to the formation of vessels is much greater in some than it is in others; (2), that in blastemata which remain in their original crude solid state, and do not break up by fibrillation, very little vascular development takes place. The vessels originating as just described, are larger than capillaries, at least in parts of their courses, less uniform in caliber, and more delicate; they communicate with the general circulation by extending through the

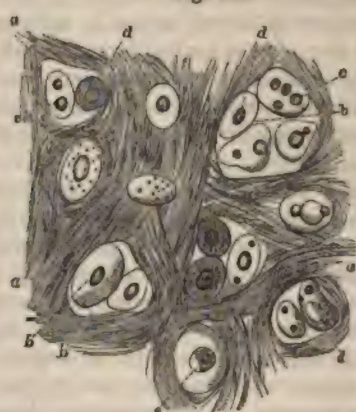
blastema till they meet with the vessels of the subjacent membrane, and anastomose with them. Another variety in the mode of development of new vessels has been observed by Rokitansky in cancerous blastemata. Parent-cells, such as might have contained a brood of young cells, were seen filled with red blood-globules; offsets proceeded from these cells filled with the same red contents, and formed anastomoses with similar ones from adjacent cells. The same special tendency to vascular development is evidently inherent in some cancerous and non-inflammatory blastemata as in the common; thus, encephaloid growths are sometimes so highly vascular as to be considered a distinct variety, the Fungus Hæmatodes; in multilocular ovarian tumors, some loculi will be found, whose contents are red with blood, while in adjacent ones scarce any trace is visible. To say why vascular formation should so remarkably predominate in one growth, or part of a growth, rather than another, is as difficult as it would be to explain why one organ, or one part of an organ, is naturally more vascular than another; for instance, why muscle is more vascular than tendon.

From these remarks upon the excessive development of vessels, we naturally pass to the consideration of growths which mainly consist of them, and are consequently called *vascular tumors*. Of these there seem to be two kinds, one consisting of dilated, arterial, or venous branches, with a certain quantity of interposed areolar tissue, to which the name *angeiectoma* may properly be applied; the other presenting a more truly *erectile* texture, *i. e.* spaces separated by intervening columns of fibrous tissue, and lined by an epithelium, which are distended by blood in a greater or less degree. The communication between the cells and the arteries is more or less free; when the former is the case, the tumor pulsates strongly. *Naevi* are tumors of this kind, occurring in the skin, which is their most frequent seat; they also occur, however, in numerous other parts, even in the interior of bones, which become dilated and thinned by their growth. They are very often congenital, their increase in size sometimes is very rapid, and seems to be favored by a contusion, or any cause of irritation. Of the non-malignant character of erectile vascular tumors there can be no doubt; they contain nothing but a fibrous structure interposed between their vessels; but with regard to some other growths, more of the character of the angeiectomata, there may often be room for doubt, at least during life. Microscopical examination, however, of the tumor, will in most cases readily show whether any elements which can be considered as cancerous are mingled with the dilated vessels. A simple vascular tumor, though it may accidentally burst, and give rise to hemorrhage, does not soften, and ulcerate, and bleed in the same continuous way that an hæmatoid cancer does. Little is positively known as to the way in which vascular tumors are formed; the angeiectoma, consisting as it does of dilated vessels, must in some way proceed from a defective contractility of the vascular coats, which yield to the distending impulse of the heart. This defect seems in some cases, as in those quoted by Dr. Carswell, to pervade the arterial system throughout, or at least in a considerable extent, instead of being localized in one part (v. fig. 3, plate iv. in his work), where the common iliac arte-

ries and their divisions are strangely contorted and expanded into pouches and dilatations. Erectile formations must acknowledge some other cause more deeply affecting the original formative process, but we do not think with Rokitansky that their cells originate as excavations in a solid blastema. The rapidity with which, when imperfectly removed, they often return again, also indicates a special modification of the laws which determine the formation of tissue.

Enchondroma.—This name was applied by Müller to certain tumors

Fig. 56.



Enchondroma; microscopic structure—after Lebert.

essentially consisting of cartilaginous structure, whose real nature was first discovered by him. They must be carefully distinguished from all

Fig. 57.

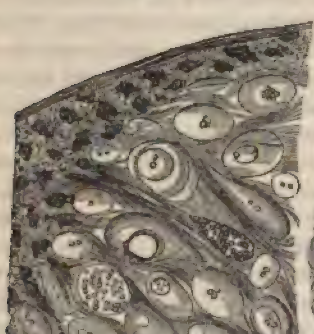


Fig. 58.

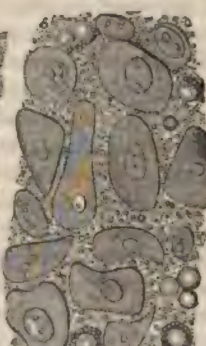


Fig. 59.

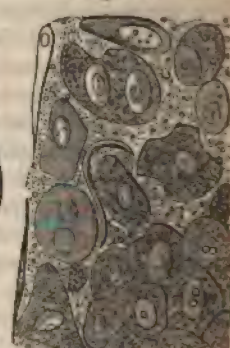


Fig. 57. Thin section of the circumference of an enchondroma from the pelvis.
Fig. 58. Corpuscles from the softened part of the same tumor.
Fig. 59. The same, after the addition of acetic acid.—Bennett.

those dense white fibrous thickenings which present very much the external aspect of a layer of cartilage. Enchondroma forms usually a

globular tumor, with a smooth or somewhat tuberiform surface. Internally, a section displays a number of loculi of very irregular roundish shape, filled with a firmly gelatinous, or rather pellucid substance. The walls of the loculi consist of a whitish fibrous tissue, more or less closely resembling that of fibro-cartilage; in some much rarer cases, this is replaced by an amorphous, firm, intercellular substance, very similar to that of true cartilage. The gelatinous matter consists of roundish or elliptic cells, varying from $\frac{1}{1800}$th—$\frac{1}{800}$th inch in diameter, containing granular nuclei, or secondary nucleated cells in their interior. Enchondroma, by boiling, generally yield chondrin, the same substance as is obtained from ordinary unossified cartilage; occasionally, however, it yields glutin. Tumors of this kind sometimes originate in the interior of the bones, which they expand so as to form a thin capsule covering their surface; this is sometimes absent entirely, or may be deficient at certain spots. In the interior of the mass there often remain inclosed portions of the cancellous tissue of the bone. Vogel states that enchondromata may originate also on the surface of bones, especially the flat; in this case they are of course invested only by periosteum, and not by any bony capsule. Enchondroma is occasionally observed in soft parts; it contains then no bone, and its structure is more purely that of hyaline cartilage. The chosen seat of enchondroma is in the bones, especially in the phalanges of the toes and fingers; out of thirty-six cases the metocarpus and phalanges were affected twenty-six times, the tibia three, the ilium one, the cranium one, the ribs one. In the four other cases, the testicle was in two the seat of the disease, the parotid in one, the mamma of a dog in one. Rokitansky mentions having seen it also in the subcutaneous cellular tissue, and in the bony. It occurs most often in the young, but sometimes has not appeared until an advanced period of life; when this is the case it is often associated with exostoses and osteophytes. Enchondroma is of slow growth, and does not usually attain a large size; one instance, however, is recorded, in which the tumor weighed nine pounds and a half. They do not contaminate the system, and only become dangerous if they inflame and ulcerate, and pour out a copious discharge. Cartilaginous tumors are prone to ossify, and in this instance the term is not misapplied, as true bony structure is really found. The ossific change sometimes seems to have commenced at the root of the tumor, at its bony attachment, and to extend outwards towards the circumference, following the progress of the cartilage formation, just as it does during the growth of the shaft of a long bone. In this way a cartilaginous tumor is converted into an exostosis. In other cases ossification commences from numerous independent centres, from which bony fibres radiate in all directions, and would, after a time, become confluent.

MYELOID TUMORS.

In his eighth lecture, Mr. Paget has described, with his usual a class of tumors with which pathologists have but recently be quainted. They are termed *myeloid*, from their being usua

loped in or upon bones, and from the similarity of their structural elements to certain corpuscles found in the marrow (μυελον) of young bones. The tumor may either be inclosed in a capsule . expanded bone,

Fig. 60.

Remarkable example of ossification of enchondromatous tumor.

or only by a periosteal investment. They are, for the most part, rather firm, but brittle, compact, of spherical or ovoid shape when invested by bone, more irregular and lobulated when growing upon bone. The cut surface is smooth, uniform, shining, succulent, with a yellowish fluid; commonly variegated by blotches of dark or livid crimson, or various shades of red upon a greenish or grayish white ground. The structural elements characteristic of myeloid tumors are large, round, oval, or irregular cells, and cell-like masses, of clear, or dimly granular substance, $\frac{1}{300}$—$\frac{1}{1000}$ inch diam.; and containing from two to ten or more oval, clear, and nucleolated nuclei. With these occur caudate and fibre-cells, and free nuclei, and the whole are imbedded in a dimly granular substance, mingled with more or less of molecular fatty matter. The history of these tumors, as far as is known, is nearly as follows: They usually occur singly, are most frequent in youth, and very rare after middle age; they are of slow growth, cause no pain, have no tendency to ulcerate, and are not apt to recur after complete removal. To the naked eye they may closely resemble malignant growths, but they have not any of their essential characters. For further details and illustrative cases, we refer to Mr. Paget's work, p. 212, from whence the above summary has been taken.

OSSEOUS TUMORS.

These may be divided into *exostoses*, *osteophytes*, and *osteoid* tumors. Before proceeding to describe them, we shall mention some of the various parts and tissues which have been observed to be the seat of true osseous formation. Articular cartilages rarely, if ever; the laryngeal and costal very frequently, especially in advanced age, and under the irritation of adjacent tuberculous ulcers, undergo ossification. The an-

terior vertebral ligament occasionally ossifies, so do the tendons, fasciæ, and aponeuroses in various parts. Dr. Walshe enumerates the following instances of the formation of bone in areolar tissue, in the submucous tissue of the gall-bladder, in the sub-serous of the pleura, the sub-retinal, the intramuscular, the parenchymatous of the liver. He mentions also ossification of muscular fibre and of the crystalline lens as having occurred.

An exostosis is an osseous tumor, proceeding from the bone or its periosteum, and, according to Rokitansky, homologous in texture, when fully developed, with that of its base and point of origin, whether that be compact or spongy. Its form varies—being sometimes broad and flat, sometimes round and prominent, with a narrow neck, some-

Fig. 61.

Fig. 61. Remarkable osseous tumor of os innominatum. Front view.

Fig. 62.

Fig. 62. Back view of same preparation.

times spinous. They do not often attain a size above that of a hen's egg. Sometimes they are single; but often several exist together in the same individual, and even on the same bone. In texture, they vary considerably from that extreme degree of density which constitutes the "ivory exostosis" to the porosity of ordinary cancellous tissue. The compact exostosis "is compact from the very first, and grows in such a way that the layers which are added to it always at once become

as dense as ivory." "When they are minutely examined, the number of peripheral laminæ is found to be very considerable, and the corpuscles lying amongst them are long. The Haversian canals are small and far apart," and the corpuscles in some parts are quite absent, in others are closely crowded together. "The spongy exostosis proceeds from a circumscribed rarefaction or expansion of the bony tissue. It forms a tumor of cellular texture, abounding with marrow, which is surrounded by a compact layer or rind." It may originate from the compact outer layer of the bone, or from its spongy interior. In some cases a tumor of this kind contains in its interior a well-formed medullary cavity, communicating with that of the bone. The spongy exostosis may remain in the same condition, or become indurated and more similar to the compact, by the deposition of more earthy substance. If, as occasionally happens, a bony tumor grows inward into the medullary canal of a bone, it is termed an *Enostosis*. Exostoses often arise without any obvious cause; sometimes they appear to develop in consequence of a blow or strain. "In most cases the periosteum covering them is in its natural condition;" sometimes it is thickened and unusually adherent. They are not peculiar to any period of life; those of the spongy kind have been observed even in new-born infants. Spongy exostoses are sometimes destroyed by caries; and in a few cases the ivory exostosis has become necrosed, and been thrown off. This change must proceed, no doubt, from obliteration of the Haversian canals, and consequent cessation of its nutrition, and would be similar to the shedding of the antlers of the stag.

Osteophytes.—To this class belong a great variety of bony growths, which form, for the most part, in inflammatory exudation, are pretty widely spread, in many cases, over a bone, and are rather easily separable from it. In these respects, and in their greater irregularity, they differ from exostoses, which are to be regarded as outgrowths from a bone, while osteophytes seem only to be produced under the influence of a bone. Osteophytes, of warty or stalactitic shape, are very common in the neighborhood of diseased joints, where the articular surfaces are affected with caries. They result, doubtless, from ossification of the exudation, derived from the adjacent hyperæmic vessels. In front of the vertebral joints and some other synchondroses, pretty long styloid or lamellar osteophytes frequently are produced, so as even sometimes to form a kind of bony capsule around them. "They arise from a chronic inflammation of the bones," and while these are atrophied, are often of dense hard texture. Sometimes the osteophyte is said to be foliaceous, consisting of beautiful delicate lamellæ, arranged parallel to each other, and running transversely vertical to the axis of the bone. The flat osteophyte is sometimes an extremely thin and delicate layer (like hoar frost); sometimes it is one or two lines in thickness. It is composed of delicate fibrils and lamellæ, and at first has scarce any attachment to the bone upon which it lies. "At a later period the osteophyte is found attached to the bone by some intervening minute round pillars and plates. After having become gradually compact, it unites with the bone." To this variety belongs the puerperal osteophyte, to which attention has of late been particularly directed. An account of this will be given in the chapter on the osseous system.

Osteoid tumors, in Rokitansky's opinion, are simply cancers, in which the stroma has undergone true ossification. This view is favored by the fact, that similar tumors are very apt to develop themselves in other and internal parts, whether the original growth has been removed or not. Lebert, however, distinguishes them from cancer, and, from the accounts which are given of their structure, not without reason. They are described as tumors of irregularly protuberant surface, sometimes of rapid, sometimes of slow growth, occasionally attaining a very considerable magnitude, and originating (the primary ones) from some bone. In structure, they consist of a cancellous bony tissue, which is plunged amid a grayish white, vascular, fibrous material, in which a sparing quantity of cells and nuclei are discernible. This substance seems to be similar to the ossifying basis of the cancellous tissue, and yields, on boiling, neither gelatin nor chondrin. The bony skeleton of an osteoid tumor may predominate more or less, so that the structure may be like compact or spongy tissue. Varieties in this respect may be observed among the different tumors in the same individual. Osteoid tumors have been found in the cellular tissues, the serous membranes, the lungs, the lymphatic glands, and in the interior of the great vessels. From these and other facts, Lebert arrives at the rather vague conclusion, that the cause of the appearance of these tumors is a general osteoplastic diathesis. This does not advance our knowledge much, and, besides, we doubt whether it touches the principal point, which is, to know whether the tumor is essentially a bony tumor, the fibrous part serving only as an ossifying basis, or whether the bony formation, though constant, is secondary and inferior in importance to that of the soft matter? Further observations must determine this. We may mention, that the name *osteo-sarcoma* is given to encephaloid tumors, originating from bone, and receiving into their mass long thorn-like or stalactitic or radiated offshoots of bone, from the natural tissue in the vicinity. These, in some cases, may resemble very much the arrangement of the bony portion of osteoid.

CYSTOID TUMORS.

These constitute a very large and important class of new formations. They agree in the one general character, that they form receptacles which are filled with various contents, but in other respects they present very great diversity. We shall separate them at the outset into two divisions, the one comprising cysts, which result from the distension of a natural pre-existing cavity, the other those which are entirely new formations. Under the first head we notice: (1.) The common so-called *encysted tumors*, which occur so often on the scalp and elsewhere. These are essentially sebaceous follicles, whose orifice has become obliterated, and the cavity in consequence, distended by continually accumulating secretion. The contents of these cysts are of very different appearance, and the names of *meliceris*, *hygroma*, *atheroma*, *gummy tumor*, have been given to express a honey-like, watery, pultaceous, or jelly-like condition of the retained secretion. Examined microscopi-

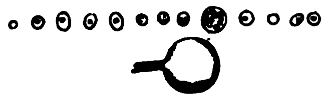
cally, epithelial scales, free fatty matter, tablets of cholesterin, crystals of triple phosphate, and small hairs in various proportions, the epithelium, however, usually predominating, are found to constitute the contents of these cysts. The cyst itself appears as a thin fibrous layer, lined on its inner side with epithelium. We have seen one case in which the epithelium in several tumors had accumulated in a very thick layer on the interior of the true cyst, giving rise to the appearance of a thick-walled cavity with contained matters of the ordinary kind; microscopic examination, however, showed that there was no real thickening of the cyst itself. The scaly particles of epithelium seem to fill themselves occasionally with a pellucid refracting matter, apparently of oily nature; they are mingled in some cases with granular globules, not unlike pus-corpuscles, or if inflammation has occurred, with pus itself. This, at least, is the case with *comedones*, which are of a similar nature. (2.) *Mucous encysted tumors;* these are essentially similar to the preceding, and are formed by obstruction of the duct of a mucous follicle, or small conglomerate gland. They contain usually a glutinous mucous fluid. They occur in the lips, the mouth, in the cervix uteri, the Meibomian glands, and in the vagina.[1] *Ranula* is an exactly analogous affection of the duct of the sublingual gland. (3.) Some of the renal, and mammary, and probably most of the hepatic cysts are produced in the same way, by local obliteration of the duct canals at two points, and distension of the intermediate portion. (4.) Single cysts in the ovary are perhaps formed by dropsical distension of the Graafian vesicles; this may also be the origin of others which are afterwards compound. (5.) Cysts in the thyroid gland are, no doubt, often formed by simple expansion of the normal vesicles. (6.) Certain bursæ (not of new formation) become distended by a persistent increase of their secretion, and constitute cystic tumors. Mr. Simon states that the contents of these, instead of being fluid, are occasionally solid, the albuminous secretion having been replaced by a fibre forming (probably fibrinous) blastema.

Under the second head, we notice: (1.) Simple serous cysts, and synovial bursæ. These arise in some cases evidently from the effect of pressure or friction, in others without any such cause. In the former case, we observe that a kind of condensation takes place in the areolar tissue of the part, making out the limits of the commencing bursa; within this the fibrous bands are gradually absorbed, while a secretion at the same time of fluid takes place, and at last the cavity is lined by a more or less perfect epithelium, and the new formation is complete. We can discern the purpose for which such cysts are formed, the end they serve, but we have no idea of the nature of the action which determines their formation. With regard to the others, which form in situations removed from pressure, as in the broad ligament of the ovary, the cause of their production is utterly unknown. They consist of a wall of fibrous tissue, varying in thickness in some measure, according

[1] We found the contents of an encysted tumor of the eyelid, operated on by Mr. White Cooper, to consist of a colorless, translucent matter, made up of multitudes of delicate granulous globules, imbedded in a clear fluid, which was coagulated in some measure by acetic acid.

to the size of the cyst, condensed so as to form a smooth surface internally, and lined by a thin layer of epithelium, which has generally appeared to us to consist of nuclear particles, with imbedding granulous matter, and not of perfect cells. The import of this condition of epithelium appears to have reference to the rapid secretory action which takes place. The fluid contents of these cysts may be poor or rich in albumen, may contain abundance of cell forms, or very few, and may be either loaded with cholesterin, or devoid of it. We can confirm the

Fig. 63.



Simple serous cyst, and epithelial particles from its interior—from vicinity of ovaries.

statements of Mr. Simon, that the granule-cells, which are sometimes very numerous, are the seat of the color of the dark coffee-ground-like matter which is sometimes present in large quantity. Some of these simple cysts contain quantities of fat, hair, teeth, and even bone, so that some good authorities have expressed their belief that they were the remains of a partially absorbed fœtus. This is certainly not the case, but their occurrence is of extreme interest, and being peculiar to ovarian growth, suggests very strongly that the normal reproductive function of this organ exerts itself by the development of these productions within its germ-bearing cavities, under the influence of some unnatural stimulus. This, of course, applies especially to the cysts alluded to under the first head, as developed from the Graaffian vesicles; but it is Rokitansky's opinion, that cysts of new formation may develop like products also. Mr. Paget states, that in these cases the wall of the cyst acquires in some part the character of true skin, with hair follicles, sebaceous, and sometimes perspiratory glands; and infers that "the structures and secretions formed on this portion of the cyst are shed into its cavity, and there accumulate; and that they remain, when, as often happens, the cutaneous structure on which they are produced has degenerated and disappeared." Several simple cysts may exist together in the ovary; this, we should consider most likely to occur when they result from development of the Graaffian follicles. Simple cysts not unfrequently occur in the mammary gland, or rather in the dense areolar tissue investing it; they have a wall of condensed fibrous tissue, and according to Mr. Birkett, are lined by a characteristic epithelium, consisting of hexagonal particles. Their contents are either limpid, opalescent, non-albuminous fluid, or a tenacious, slimy, opaque, variously colored, and concentrated solution of albumen. When combined with a peculiar growth of gland tissue advancing into their cavity, these tumors constitute the sero-cystic sarcoma, of which we shall speak more particularly when we come to the morbid anatomy of the mammary gland. Cystic formation may take place in various kinds of tumors, in fibrous, carcinomatous, sarcomatous, and may be a more or less prominent phenomenon. (2.) *Compound cysts.* The chief seat of

these is in the ovaries, where they present two principal modes of development. In one of these the parent cyst, which, for the most part,

Fig. 64.

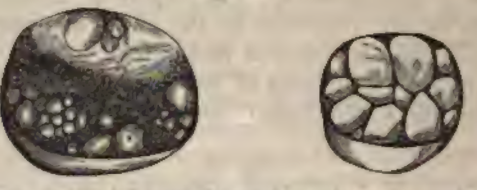


Diagram of compound cysts. In the left figure, the secondary cysts are seen growing on the inner surface of the parent. In the right, they have filled up the cavity.

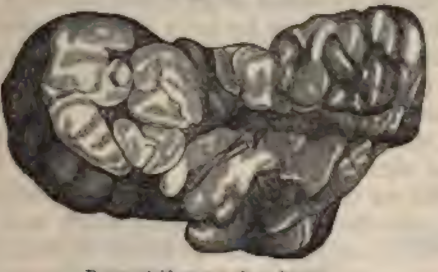
continues to predominate in size, gives origin to a second generation of cysts, and then again to a third, and so on. The consecutive series of cysts are developed in the walls of their parents, but do not grow inward and occupy their cavities; the result is a multilocular mass, made up of numberless cysts, which are filled with very various contents. These may be tolerably limpid and clear, or very viscous and greenish; may contain a very large number of celloid particles, or very few; may be variously colored by blood-globules of new formation, or even replaced by a solid blastema loaded with developing blood; fat, hair, teeth, and bone may also occur in these, as well as in the simple cysts. The partitions between the various cysts sometimes give way, and thus a tumor is produced, which internally seems imperfectly divided into compartments. In the other mode of development, the secondary cysts grow inward into the cavity of the parent, which they fill up more or less completely, a tertiary race behaves toward them in the same way, and so on. This form may be combined with the preceding. Rokitansky describes a kind of villous, or cauliflower growth, which originates on the wall of the secondary or parent cyst, and may increase so as not only to fill the cyst cavity, but to break through its wall, and vegetate in the cavity of the peritoneum.[1] The impression left on the mind of the observer after a minute examination of the compound cysts, is, that they are of the lowest type of organization, resulting, apparently, from a depraved, degenerate formative action, which, withdrawing blastema from its proper uses in the system, hurries it with a wasteful expenditure into useless and injurious elementary shapes. How precious is the stringency of the law of our organic constitution, which is comparatively seldom infringed by such terrible aberrations! It may be remarked, that the tendency to cyst formation most often appears in the existence of several together; it is certainly far more common to find several, whether of new formation, or resulting from distension of natural cavities, than to find a solitary one. This indicates some special modification of the normal organic action; but we cannot think it is such as Rokitansky points out, when he ascribes all cyst formation, ex-

[1] In the Report of the Pathol. Society for 1851–52, there is an account, at p. 404, of a growth on the interior of an ovarian cyst which seems to be of this kind. It consisted of "vast numbers of pedunculated, clavate, clustered growths, formed apparently of a simple basement membrane inclosing cells."

cept that arising from mere distension, to the extraordinary development of a primary cell.

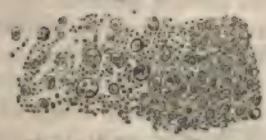
Sarcomatous tumors constitute a group, which, it must be allowed, is very ill defined. We may describe what we regard as the characters of a typical specimen; but we shall seldom find them all present in any given instance; and, frequently, they will approximate so closely to those of other classes, that we shall remain in some degree of doubt. The fibrous group on the one hand, and the carcinomatous on the other, are the territories which border on the debatable land of sarcoma. The characters attributed to sarcoma are the following: It occurs as a local, for the most part solitary formation, not affecting the constitution. If removed completely, it does not return. In shape it is well defined, roundish, with uneven or lobulated surface, often also branching and

Fig. 65.



Pancreatoid sarcomatous tumor.

Fig. 66.



Structural elements of same.

extending itself between the adjacent tissues. Its size varies from that of a hazel-nut to that of a cocoa-nut (Walshe), or perhaps may be still greater. It seems to have no particularly determined site; both maxillæ (especially the upper), the interior of bones, glands, muscles, fibrous membranes, submucous areolar tissue, even the brain, are all mentioned as being affected by it. It belongs to an earlier period of life than carcinoma. Softening and breaking down do not necessarily occur in the progress of sarcomata; but if they are exposed, by sloughing of the parts which cover them, they may inflame and *saniate*, or slough.

The commonest variety of sarcoma is that which Rokitansky calls the *albuminous fibrous tumor*, on account of its yielding, on boiling, no gelatin, but an albuminous matter. The fibres are of various kinds, scarcely different from those of ordinary fibrous tumor. They lie in a diffused albuminous blastema, and are interwoven with a more or less abundant vascular plexus. Another variety is the *gelatinous sarcoma*, the same which Müller has named "collonema." It is sometimes very

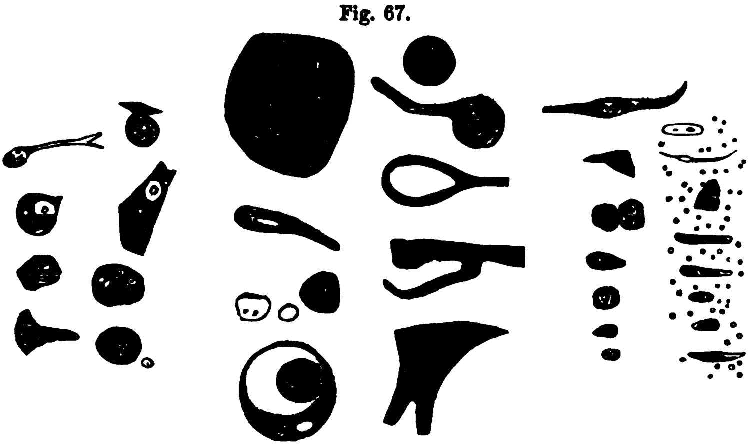
soft, clear, tremulous, like jelly, scantily supplied with vessels. In structure it presents, in different specimens, varying proportions of fibres, white or elastic, of celloid particles, elementary granules, and fibrillating blastema. In other instances the structure is more firm and resistent, of lobulated aspect, consisting of white filamentous tissue, intermingled with caudate nuclei and cells. In others, again, the inter-cell substance is firmer, more stiff and amorphous, so that the structure approaches that of enchondroma. Some sarcomatous tumors present a not very distant resemblance to the conglomerate gland structure, their mass dividing into lobes and lobules. To such, the name of pancreatic sarcoma has probably been applied. It is, however, pretty certain, that the same kind of structural arrangement exists in some cancerous growths. M. Lebert has examined sarcomatous tumors very carefully, and designates them as fibro-plastic. By this term, he seems to imply that they constitute a transition stage towards fibrous tumors; and the excellent detailed descriptions he gives of their structure, are, on the whole, confirmative of the same view. Nuclei, circular, and elongated cells, and fibres, make up the chief part of their structure—the fusiform cells being generally the most numerous. It is to be regretted that the tumors he examined were not tested, so as to ascertain whether gelatin could be obtained from them in quantity. If this had been the case, their fibrous character would have been decided. Among the tumors we have ourselves examined, those which seem to deserve best the name of sarcoma, as being unlike either to fibrous and carcinomatous growths, are certain enlarged lymphatic glands, whose structure consisted of multitudes of nuclei, set in a fibroid stromal substance. With these we should class certain separate tumors, entirely new formations, which are rather soft or lax, of a whitish gray or light pinkish color, either smooth on the surface or lobulated, so as to resemble the pancreas. In structure, these are found to consist of myriads of nuclear, with a few celloid, particles, and some trace of fibroid stroma mingled with a large proportion of faintly granulous matter, and more or less oily. These tumors contain notable quantities of gelatin and protein. Generally, we should conceive that a growth, consisting of cells or of nuclei, set in a non-fibrillated homogeneous blastema, would not yield gelatin, but albumen; while one which consisted of fusiform cells or fibres, would have more or less completely undergone the chemical change which an albuminous blastema experiences in passing into the state of gelatinous fibre. The former, we should, therefore, consider to be properly termed a sarcoma, *i. e.* a simple growth, of like composition to flesh, or the albuminous blastema, effused from the blood, not having attained in its development any very special structural character. In proportion as it passed into the condition of a fibrous tumor, it would lose its sarcomatous character. Cyst formation, combined with sarcoma, constitutes *cysto-sarcoma*. This is said by Rokitansky to occur in three forms: (1) *simple cysto-sarcoma;* (2) *cysto-sarcoma proliferum*, in which young cysts grow on the inner surface of the parent, and are either sessile or pedunculated; (3) *cysto-sarcoma phillodes*, distinguished by a growth of vascular, laminated, or watery or cauliflower-like excrescences projecting into the interior of the cyst. The structure of these growths

is sometimes more fibrous, sometimes more similar to that of gland tissue. In both cases, the foliated terminal portions are invested by a distinct limitary membrane, within which an epithelium is sometimes discernible. Cysto-sarcomata mostly occur in the female breast and in the ovary.

CANCEROUS TUMORS.

In attempting to give a sketch of cancerous or malignant tumors, we think the best plan will be to take a typical specimen, which presents all the characters of the genus strongly developed, and to point out what these characters are. We shall afterwards notice the several species, and endeavor to show how the distinguishing features gradually become effaced, until the formation, as often happens, is, or appears, almost identical with those of a benignant nature. A tumor, of the species called encephaloid, is certainly the παράδειγμα of cancer. It is of rapid growth, often attaining in a short time a very large size. Its

Fig. 67.



Encephaloid.—The first and the last three of the sets are from the liver, the second is from a bone, and the third from the vertebral column. The great difference of the cell forms is very apparent.

aspect, resembling very closely that of the medullary cerebral structure, has obtained for it the name just mentioned, as well as others of similar import—*medullary sarcoma*, *medullary fungus*. Its color is an opaque white, often, however, varied in parts by patches of deep red, from vascular injection or hemorrhage. Its consistence is often so soft, that it seems semi-fluid, and gives the sensation of fluctuation. In structure, it appears on section often almost homogeneous, sometimes with a locular or fibrous arrangement. Microscopic examination shows that the main mass consists of celloid particles and cells, contained in a sparing quantity of filamentous tissue, which forms a kind of stroma. Such a tumor is seldom solitary, but coexists with other similar ones in the same and in different organs. If extirpated, it is sure to return,

and probably diffuse itself more widely than before. It poisons the lymphatic current passing from it, and induces growths of like nature

Fig. 68.

Simple and compound cancer cells from cancerous duodenum.—Bennett.

in the glands which that current traverses. It affects the general system with a peculiar cachexia, marked by languor, emaciation, debility,

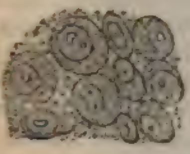
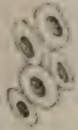
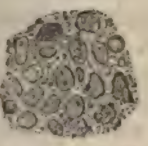
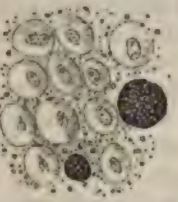
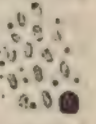
Fig. 69.

Cells from Encephaloid of Tongue (rapidly growing).

and a sallow complexion. It is very apt to infiltrate adjacent textures with its own substance, and, by absorbing their nourishment for itself, to occasion their atrophy; and, lastly, it tends, when exposed, to break

down by a kind of decay, and to pour out profuse, exhausting discharges of serous, sanious, or bloody fluid. Such is cancer in its most malignant form. Of the above-mentioned characters, those which seem to us most nearly pathognomonic, are the tendency to infiltrate adjoining parts, to affect the glands traversed by the issuing lymph-current, to reproduce similar growths in distant parts, and to return after removal. If these

Fig. 70. Fig. 71. Fig. 72. Fig. 73. Fig. 74.

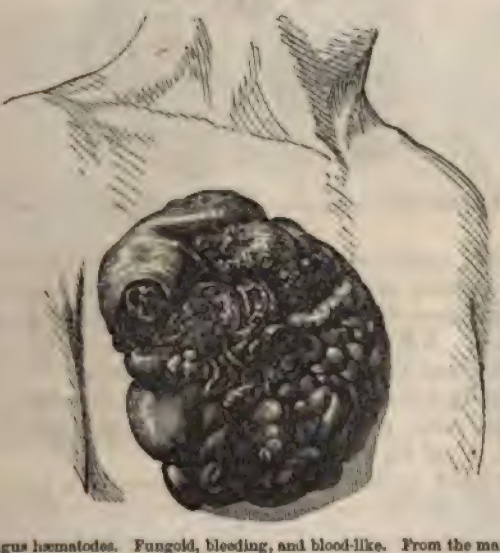






Figs. 70, 71, 72. Cancer-cells before and after the addition of acetic acid, also the structure of the reticulatum from encephaloma of the testicle.

Figs. 73, 74. Young cancer-cells before and after the addition of acetic acid.

four characters are decidedly exhibited by any tumor, there can be scarce any doubt of its malignant nature. It is to be remarked that all these characteristics are dynamic and not structural. They result from the invisible qualities of the new formation; its mode of vegetation, dis-

Fig. 75.

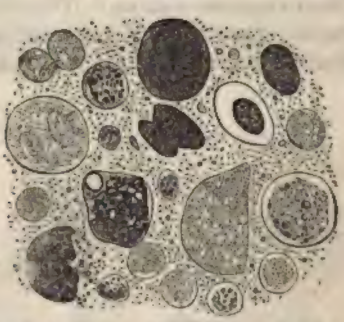


Fungus hæmatodes. Fungoid, bleeding, and blood-like. From the mamma.

semination, and reproduction, not from any peculiarity of form or arrangement of its particles. Whatever these might be, a tumor, which behaved as we have just described, would proclaim its cancerous nature.

This point we shall illustrate further on. We now proceed to speak of the other varieties of cancer; first, however, noticing the sub-varieties of encephaloid. The name *mastoid* is given to a kind of firm growth, which is thought to resemble on section the boiled udder of the cow. That of *solanoid* (potato-like) designates other hard cancers, resembling that vegetable when sliced. Dr. Walshe says they are a pale yellowish, of unctuous crisp look, and almost homogeneous. *Milt-like* has evidently reference to a soft, pale growth, containing scarce any blood. The surface of a section of *Nephroid* cancer presents a resemblance to that of a kidney, owing to the peculiar arrangement of its fibres, which are themselves of a delicate gelatinous transparency, so that Rokitansky calls the growth hyaline cancer. The term *Hæmatoid* expresses a much more important feature than any of the preceding, as it implies that the growth is unusually vascular, is the seat of excessive development of vessels, and perhaps of blood, and is prone to pour out those alarming hemorrhages which often cause fatal exhaustion. When the hæmatoid character is strongly marked, the name of Fungus Hæmatodes is applicable. *Melanoid* cancer is, in the great majority of cases, encephaloid structure, with the addition of black pigment. The cell-growth of

Fig. 76.



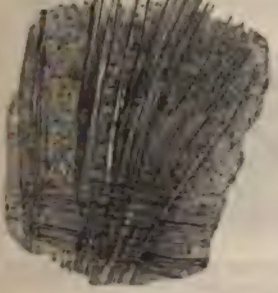
Cells more or less loaded with black pigment, from a melanotic tumor of the cheek.—Bennett.

encephaloid may consist of large free nuclei, of caudate cell-particles, of granulous globules much like those of pus, but unaltered by acetic acid, of cells of most various shapes, often irregularly caudate, and of pellucid vesicles. All these may be mingled in various proportion, or some may constitute the chief mass of a growth. The annexed cut, page 187, exhibits cell structures of various kinds; and for more minute details we must refer to the trustworthy descriptions of Mr. Paget, page 368, vol. ii., of his published lectures.

Scirrhus or hard cancer commonly appears as a knotty, or uneven, pretty distinctly limited, very hard (stony) tumor. Its surface or section is of a bluish or grayish white, and often presents a peculiar glossiness; scarce any trace of vessels is ordinarily visible, except in spots, which are inflamed and softening. The adjacent tissues, especially the skin, when the growth is subcutaneous, are more or less involved, and drawn inwards towards the tumor. In structure, it consists essentially of

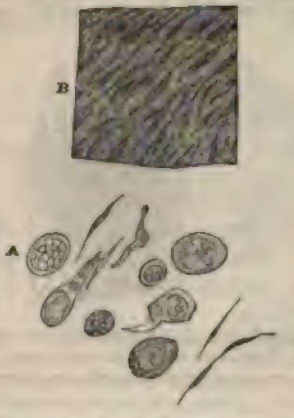
a blastema, or basis substance, more or less advanced in fibre-development, in which very various forms of cell-growth are imbedded. Of the latter,

Fig. 77.



Fibroid stroma of a scirrhous tumor of Pylorus.

Fig. 78.



Scirrhous tumor of cerebrum.
A—Cells.
B—Section of firm stroma.

it is utterly impossible to give any general account, except to correct the common idea that they are of fusiform shape, they may be bare

Fig. 79.

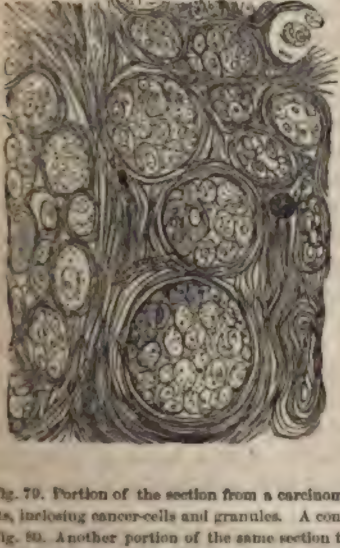
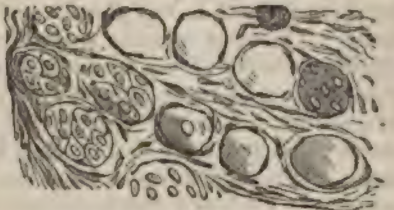


Fig. 80.



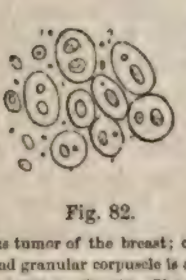


Fig. 82.

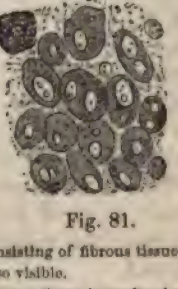


Fig. 81.

Fig. 79. Portion of the section from a carcinomatous tumor of the breast; consisting of fibrous tissue and cysts, inclosing cancer-cells and granules. A compound granular corpuscle is also visible.

Fig. 80. Another portion of the same section treated with acetic acid. The fibrous tissue is rendered more transparent, and elongated nuclei are visible scattered through it. The nuclei of the cancer-cells are unchanged, while their walls are transparent. A compound granular corpuscle is seen at the upper part of the figure.

Fig. 81. Cancer-cells from the cream-like juice squeezed from the tumor. Numerous granules, and a compound granular cell, are seen.

Fig. 82. The same, after the addition of acetic acid.—From Bennett.

nuclei, cells of most various aspect, vesicles; granular globules; with these oil and diffused granulous matter are mingled in varying quantity. Glomeruli are often seen in fattily degenerating, or in inflamed parts; and parent cells, containing a secondary generation, are occasionally present. The disposition of the fibres is very various, sometimes parallel

Fig. 83. Fig. 84. Fig. 85.

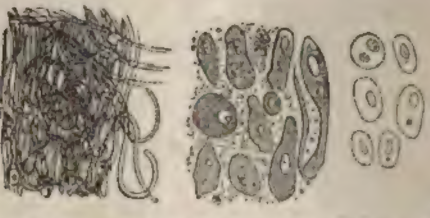


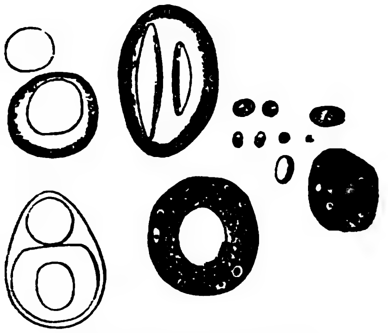
Fig. 83. Dense fibrous and elastic tissue, in which cancer-cells are infiltrated from cancer of rectum.
Fig. 84. Cancer-cells scraped from the surface, in the same case.
Fig. 85. The same, after the addition of acetic acid.—From Bennett.

to each other, sometimes radiating, often crossing at right or acute angles. An alveolar arrangement sometimes exists; it proceeds, according to Rokitansky, from the development of the parent cell. Scirrhus yields some gelatin on boiling; less albumen and oil, but more saline matter, are contained in it than in encephaloid. The growth of scirrhus is slow, the more so in proportion as its fibrous element predominates; it may then exist long without inducing the constitutional cachexia, or reproducing itself in any distant part, or even affecting the lymphatic glands. The most common seats of scirrhus are the female breast, the pyloric extremity of the stomach, the rectum; it is usually the original formation in these or in other parts; but gives rise to secondary encephaloid growths. A tumor of this kind does not, even when most defined, possess a true cyst; often, it extends itself by infiltration among adjacent tissues. It rarely attains a large size, it is not often seen so large as an orange. Various names have been given to some peculiar appearances occasionally presented by scirrhous tumors; of these, we shall only mention the *napiform*, applied to certain tumors whose cut surface presents a number of concentric lines, resembling those seen in the interior of a turnip; and the *apinoid*, or *reticulated*, in which spots or streaks of an opaque oily matter are apparent upon a grayish field, and thus occasion the appearance of separate patches, or of a network. It is ascertained that this latter condition indicates a commencing fatty change. The surface of an ulcerating cancer is irregular, of a grayish or faint reddish aspect, covered with a thin watery sanies, or with a layer of sloughing detritus; the margin of the surrounding skin is commonly elevated and everted.

Colloid cancer, the next variety we notice, is also called alveolar; other cancers may present more or less of an alveolar arrangement, but it never constitutes the prominent feature of their structure. The walls of the alveoli consist of a fibroid tissue, sometimes extremely delicate and translucent, sometimes, and especially in the deeper layers,

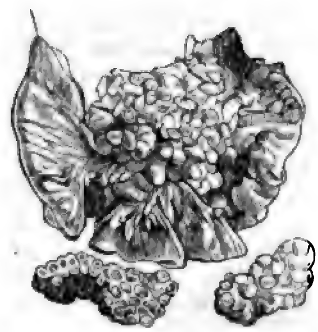
strong and firm. The contained loculi vary in size from that of a grain of sand to that of a pea; they are round or oval; occasionally adjacent

Fig. 86.



Colloid cancer of a lymphatic gland.

Fig. 87.



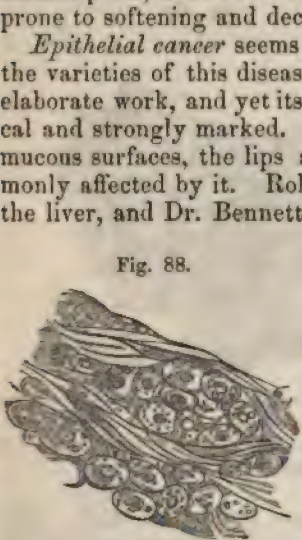
Of the two smaller figures, one exhibits the circular loculi as they appear on a section; the other shows the compound spherical character of the malignant growth itself.

ones communicate together by solution of the interposed wall. The jelly-like substance in their cavities is of a greenish yellow, semi-transparent, and clammy; "it yields no gelatin on boiling, but seems to consist of a peculiar substance, identical with that naturally occurring in the cavities of the thyroid, and in some cysts. Cells, nucleated and non-nucleated, caudate, and fusiform, nuclei, and elementary granules, occur in this substance, and, under circumstances probably connected with softening changes, granule-cells, and fat-molecules. Endogenous production of the cells within parent-cells is sometimes observed. Colloid may present itself as a distinct solitary tumor," or may infiltrate the tissues which it infests, when it occurs on serous membranes; there are often small scattered nodules of the growth in the vicinity of the larger. Rokitansky mentions the two following varieties of colloid: (1.) The contents of the loculi increase, so that their septa are in great measure atrophied and lost, and the mass presents the aspect of a tremulous jelly. (2.) The superficial alveoli enlarge considerably, and attain a prodigious size. The favorite *habitat* of colloid is the stomach and omentum; it also occurs in the ovaries, the bones, the kidneys, the uterus, and the spleen. Its growth is often rapid, and it may attain a very large size, exceeding that of a cocoa-nut. The contents of the alveoli are sometimes of pearly aspect, probably from the presence of cholesteatomatous matter. Cruveilhier has described an *areolar pultaceous* variety, in which the loculi contain an opaque, yellowish, tallow-like matter, having the chemical constitution of casein. This we believe, with Rokitansky, to be a condition in which fatty transformation of the gelatinous matter is taking place analogous to that noticed in reticular scirrhus. Colloid may exist combined with scirrhus, and also with encephaloid; in the latter case, the superficial loculi become occupied by soft encephaloid matter. The malignant

character is less marked in colloid than in the other kinds of cancer; it does not induce such marked cachexia, does not reproduce itself in distant parts, does not contaminate the lymphatic glands, and is less prone to softening and decay, or to inflammation and saniation.

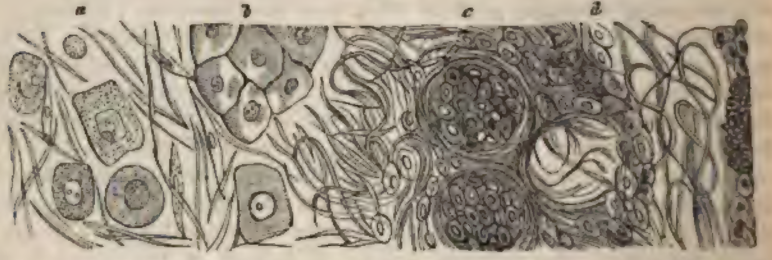
Epithelial cancer seems only recently to have been admitted among the varieties of this disease; it is not mentioned by Dr. Walshe in his elaborate work, and yet its cancerous nature in many cases is unequivocal and strongly marked. It occurs almost solely on tegumentary or mucous surfaces, the lips and cheeks are among the parts most commonly affected by it. Rokitansky mentions having once observed it in the liver, and Dr. Bennett has met with it as a secondary growth in the lymphatic glands. On mucous surfaces it appears as a cauliflower-like growth, of a more or less red tint from vascular injection, of various degrees of consistency, and easily separated into parts by pressure. On the general tegument, its appearance is most often that of a low, tolerably well-defined tumor, of hard feel, having an irregular nodulated surface, covered with minute watery papillæ; when ulceration and softening take place, the surface becomes injected, a watery and serous discharge is poured out, and gradual destruction of the part proceeds. In structure, these tumors essentially consist of an alteration of the integument, the corium and subcutaneous

Fig. 88.



Epithelial cancer.

Fig. 89.

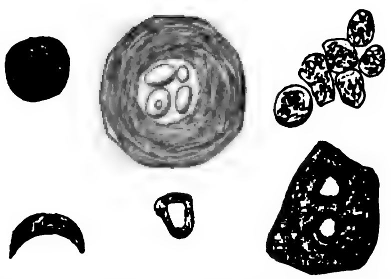


Appearance of section of cancerous tumor of the cheek. *a*. Epidermic scales and fusiform corpuscles on the external surface. *b*. Group of epidermic scales. *c*. Fibro-elastic tissue of the dermis. *d*. Cancer-cells infiltrated into the fibrous tissue, and filling up the loculi of dermis. (From Bennett.)

areolar tissue being converted into a fibroid substance, the papillæ greatly hypertrophied, as well as the epithelium resting upon them. In the last specimen we examined, which was from the lower lip, a vertical section displayed an external whitish layer, about one-third inch thick, marked by vertical striæ, and resting upon some areolar tissue, fat, and muscle. Its surface showed but slight traces of subdivision. Its extent in depth appeared to be most accurately limited by the lower margin of the whitish striated layer, but, upon examination, the areolar tissue immediately subjacent, and for some depth, was found thoroughly

infiltrated with nuclei and granular matter. Sections of the altered integument showed papillary elevations, completely overwhelmed and blended together by an enormous growth of scaly epithelium, which, in some parts, showed a tendency to fatty change, and here and there the capsulating arrangement mentioned below. If glands exist in the part affected, their epithelium may also accumulate within their canals, and thus add to the size of the tumor. One peculiarity is very commonly observable in the arrangement of the cells of epithelial cancer, which does not seem to occur in other growths; this is, that here and there the scaly particles are arranged in lamellæ around a central circular space, which appears to be a largish cell, containing a younger cell-growth. The malignant character of epithelial cancer is manifested in its extending from the superficial textures first involved to the deeper seated, even to the bones; the laryngeal cartilages have often been involved by it. It seems, however, to have less tendency to contaminate the lymphatic glands, and the system generally, than other varieties of cancer. Rokitansky describes a variety of cancer, which he calls *villous*, from its consisting of a kind of delicate fibrous stalk branching at its end into villous processes, with somewhat bulbous terminations. These contain encephaloid substance, and are extremely vascular. Hemorrhage often takes place spontaneously from them, and is easily excited by the slightest lesion. The only specimen at all corresponding to this which we have examined, was one of cauliflower excrescence of the uterus: it was not in a suitable state for accurate

Fig. 90.



Epithelial cancer—four of the loculi are shown, and some of the flattened cells, one of which is curved, having probably been arranged round a loculus.

investigation, but we saw that it consisted in great measure of large vessels, covered with a thick layer of lowly organized cell-growth. Rokitansky mentions a case of this kind, in which the growth sprang from an evidently encephaloid base. A cystic growth sometimes occurs in combination with one or other of the species of cancer, chiefly with encephaloid: the cysts may be simple or compound. The cysto-carcinomatous growth is usually of large size. There may be, probably, other varieties of cancerous tumors, or, to put it otherwise, tumors possessing more or less of cancerousness; but we have now sketched the outline of the principal forms that are usually met with, and we feel convinced that it is far more important

for the student and the practitioner, to contemplate steadily the great characteristics of cancerous disease, than to load his memory with details of the incidental and trivial. Partly on this account we have not attempted to give any very minute description of the structure of cancerous tumors, for our own examinations have most thoroughly convinced us of the non-existence of any special structural character, absolutely and in all cases distinctive of cancer. This point, which is in accordance with the teaching of the best authorities, seems far from being correctly understood in the present day, and we cannot but think that there is still much tendency to over-estimate the microscope as a means for the diagnosis of cancer. It is our opinion that the cases are very rare indeed, where the microscope will avail to detect cancer with any certainty, where the naked eye features are insufficient. On the other hand, we have more than once seen unquestionable cancers made up of substance which we should have been led, from microscopic examination alone, to consider as of a simple nature. What may be said relative to the distinguishing of cancerous from other tumors, by their mere physical characters, and not by their living actions, amounts to this: If a tumor, on being incised and compressed, yield a whitish, milky juice, (the so-called "*suc cancereux*,") it is probably malignant; we have, however, failed to obtain this sign from actual encephaloid. If the cell-growth of a tumor is what may be called exceedingly *multiform*, *i. e.* one particle unlike another, the field of view being filled with other varieties of shape and construction, there arises a strong presumption that the structure is malignant. If a tumor consist of an abundant cell-growth lying in a basis substance of slight consistence, and containing very little fibre, it so far bears a close resemblance to encephaloid. If, on the other hand, a tumor consist chiefly of fibre or fibrillating blastema, the presumption of its cancerousness diminishes; we have, however, seen a growth in the liver, which had all the aspect of a scirrhous formation, and probably was so, which yet consisted solely of fibre-forming solid blastema. If a tumor infiltrate adjacent parts, it is probably malignant, but all cancers have not this character. The presence of large cells, containing several nuclei, similar to those figured by Lebert[1] and Bennett,[2] would be a strong argument for the cancerous nature of the tumor, from whence they proceeded. So also we should regard the development of a nucleus into a large granulous globule or vesicle, or into any structure very dissimilar to its original condition, or that of the nuclei of natural tissues. In concluding these general remarks, we may state, we think, the following position with some confidence, viz: that, starting from encephaloid as the representative of cancer *par excellence*, we find the cancerous character gradually declining as we pass through a series of formations, such as we have above described, until we come to those of whose innocent nature there is no question. The exact limit, we believe, at which cancerousness is lost, cannot be marked by any characters of a growth itself. The vessels of cancer, for the most part, we believe, are of the ordinary kind, derived from those of the natural tissues by the process

[1] Phys. Patholog. Pl. xxi. fig. 5.
[2] On Cancerous and Cancroid Growth. Figs. 69 and 117.

of extension or growth; sometimes, however, it seems that blood and vessels are formed in the blastema of a tumor, as we have described them to be in exudation matter; this we consider is most likely to be the case in growths of the hæmatoid character. The blood contained in the developing vessels is seen, as it is said, to oscillate in them before they have anastomosed with those of the general system. No special formation of lymphatic vessels or nerves, seems to take place in cancerous tumors. The lymphatic vessels of the part affected, no doubt, act as absorbents of the redundant blastema, as is amply shown by the special contamination of the glands, to which those vessels immediately proceed. The nervous filaments traversing or distributed to the part which is the seat of the cancerous growth, are often involved in the mass, and becoming injuriously pressed on, or otherwise injured, occasion the most frightful pains. There is scarce anything accurately determined, respecting the chemical composition of cancer. Encephaloid is said to consist chiefly, if not entirely, of albuminous matter; Scirrhus to contain gelatin also, while colloid jelly seems to be a principle quite *sui generis*. Possibly, there may be some special cancerous virus, as there is a variolous and syphilitic, but as yet chemistry knows nothing of it, and we only infer their existence from the effects they produce.

[The subject of cancer and its microscopic diagnosis being one of deep interest, we present here the illustrations of cancerous structure from the valuable paper of Dr. Francis Donaldson, of Baltimore, Maryland. See *Am. Journ. of Med. Sci.*, vol. xxv. p. 43.

"It is improper," remarks Dr. D., "to attempt to divide cancer into so many species, as they all have the same common pathology. The variety of aspect, consistence, volume, coloration, and vascularity, is caused merely by the amount of fibrous element, of fat, or of gelatinous fluid present; all of which are purely accidental, and in no way essential to constitute the growth. The density, softness, &c., may also vary according to the organ involved; the breast and the pylorus take generally the form of scirrhus; whereas the bladder, the kidneys, &c., are more likely to be affected with encephaloid. Compare the physical characters of cancer with those of the simple tissues, such as the muscular, areolar, dartoric, osseous, &c., or with those of the compound, as the glandular, the synovial, the mucous, &c., and the difference will be very apparent. Its greater or less firmness, its homogeneous fibrous aspect with its lactescent infiltrated juice, are very characteristic. The presence of this peculiar fluid is of itself a point of differential diagnosis of great value; the microscope always detecting in it, when found, the presence of cancer-cells, &c. No matter what organ is the seat of the disease, this fluid can generally be scraped from the cut surface, or squeezed out by gentle pressure. It is particularly abundant in encephaloid, and frequently oozes out in drops having a white cloudy appearance of the consistence of cream, and very much of its color, being slightly tinged with yellow. It may sometimes, on superficial inspection, be confounded with light-colored pus, which has, however, with the yellow, a slightly greenish tinge. If, from the conditions of its formation, there can be any doubt, an appeal to the microscope will at once settle it by giving us the characteristic pus-globule. (See Figs. 99 and 100.)

"The cancer juice forms readily an emulsion with water, and in this differs from tubercular matter and from that pressed from sebaceous tumors. The color of this juice is of course modified by the mixture of other fluids with it; thus, when the vascularity is great, it is often reddish; when from a deposit of dark pigment, we have what is called melanotic cancer, it becomes of a dark brown. When mixed with much fat, it is more consistent; in colloid, it is thicker and sometimes grumous."

"In the accompanying figures, we have," says Dr. D., "attempted to arrange (under several divisions), into groups, the different forms of the cancer-cell we have met with. In making the selection from the numerous drawings we have collected in our album, we have thought it better, instead of giving only the types, so to speak, of the several shapes under which we desired to include all the various modifications, to show as many as possible of the numerous varieties. For the rudeness of the designs themselves we ought, perhaps, to apologize, but they are, as far as we could make them, exact representations of what we saw in the field of the instrument. We will first describe the proper elements separately, and then speak of the objections offered by Dr. Bennett, and some others, to their distinct characters as pathognomonic of cancer, giving drawings of other elements confounded with them. The points of dissimilarity we will call attention to with a view of fixing the differential diagnosis. The mode we have employed has been simply to place between two pieces of glass a drop of the juice, obtained either from gentle pressure, or by scraping the cut surface with a scalpel, diluted with a little water. The cutting off of small slices with Valentin's knife, and examining the whole mass together, will exhibit, almost invariably, more or less fibrous structure, but necessarily the lens employed must be much feebler, and the cell is not seen to the same advantage; moreover, the fibrous element is purely accidental, and is found in a vast number of tumors. The instrument used is a first-class one, manufactured by Nachet. The power we have habitually used in studying cancer element has been one of 555 diameters (Nachet's No. 6). Mr. Bennett used, in his researches, one of 250, which he recommends to others. We state this for the purpose of explaining why it is he has omitted some characters of the element which we believe are of great importance. The element of cancer consists of three parts, *cell*, *nucleus*, and *nucleolus*, all of which are peculiar to it. We will consider—

"1. The cancer nucleus, as inclosed in a cell, or as floating free by itself.

"2. The polygonal, or more or less spherical and ovoid cell.

"3. The caudated cell.

"4. The fusiform cell.

"5. The concentric cell.

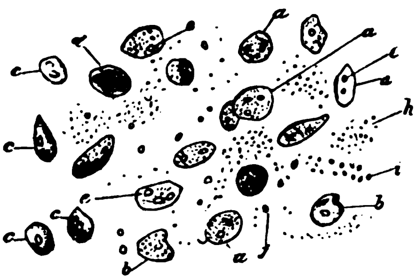
"6. The compound, or mother cell.

"7. Agglomerated nuclei connected by amorphous tissue.

"In all the varieties of cancerous tissue, nuclei are to be found either enveloped by a cell, or floating free, generally more or less of both; in some specimens, there exists a large number of free nuclei with only an occasional cell. The form and appearance of these nuclei is the most

constant and unvarying of all cancer elements. They are, Fig. 91, *a*, ovoid, or more or less round; the latter are found more particularly when the eye or the lymphatic glands are the organs diseased. Some-

Fig. 91.



Free cancer nuclei. *a*. Type form. *b*. The same, with a piece nicked out of the side accidentally. *c*. Shows a free nucleus, in which the molecular granules are very minute, often met with in perfectly fresh specimens. *d*. A nucleus, in which larger granules have commenced to form. *e*. The characteristic nucleolus with its dark contour and bright centre. *h*. Fine molecular granules. *i*. The second variety of granules, or gray granulations. *j*. Fat granules.

times (as in *b*), we find little pieces of the wall of the nuclei apparently nicked out; but evidently it is purely accidental, and the proper shape can easily be recognized. They have, ordinarily, in width, a diameter of from 1-100th of a millimetre, or (a millimetre being equal to .039th of an inch) of .0039th of an inch, to 1-66th of a millimetre, in one instance we met with one as wide as 1-38th of a millimetre; in length they measure from 1-133d to 1-100th of a millimetre. Their contour is dark and well defined, with the interior containing very minute dark granulations; indeed, when the specimen is perfectly fresh, they have a homogeneous aspect, the granulations being so small as to give the appearance of a mere shading (as in *c*); if the specimen is kept a day or two, we find the interior filling up with larger granulations (as in *d*). Within these nuclei, when they have not been obscured by granular or fatty degeneration, there is found habitually a small body, or *nucleolus*, averaging in diameter about 1-500th of a millimetre. These nucleoli have somewhat of a yellowish tinge, with a brilliant centre and dark borders, refracting light like the fat-vesicles. We would call attention, particularly, to the peculiar brilliancy of the centres of these nucleoli, which, we think, is characteristic; it can be almost invariably noticed, if the focus is varied. Their large size, in proportion to the nuclei, should also be noticed, together with the great variableness of their position, sometimes being near the centre, and again in close contact with the walls (see *e*). Ordinarily, in other elements, they are found almost constantly in the centre. Very frequently, two or three nucleoli are found within the same nucleus. M. Robin[1] mentions the action of acetic acid upon cancer nuclei and their nucleoli, as differing from that on other elements, particularly epithelial; it renders the nucleus gradually paler, together with the cell, destroying neither—but

[1] MS. notes of his Cours de Histologie, 1850.

the nucleolus is perfectly untouched by it; whereas in epithelial cells, where generally in those of the skin the nucleoli are wanting, the action of acetic acid destroys the cell, leaving the nucleus unaltered."

"It is of primary importance for the proper examination of the cancer nucleoli that the specimen should be fresh. Such being the case, we do not remember ever having found these peculiarities wanting."

"We have examined some specimens in which free nuclei were in great abundance, and where, after long-continued diligent search, we were unable to discover any cells. More particularly is this the case in cancer of the liver, of the pylorus, and of the lymphatic glands; more rarely in that of the eye. In the breast, many full-formed cells are found with more or less of free nuclei floating in the blastemic fluid. It may be well to remark here that we find also free nuclei of fibro-plastic and epithelial cells, of the finest bronchial ramifications, each with their peculiarities. Mr. Bennett appears to us to have confounded them all together in speaking of what he calls fibro-nucleated tissue.

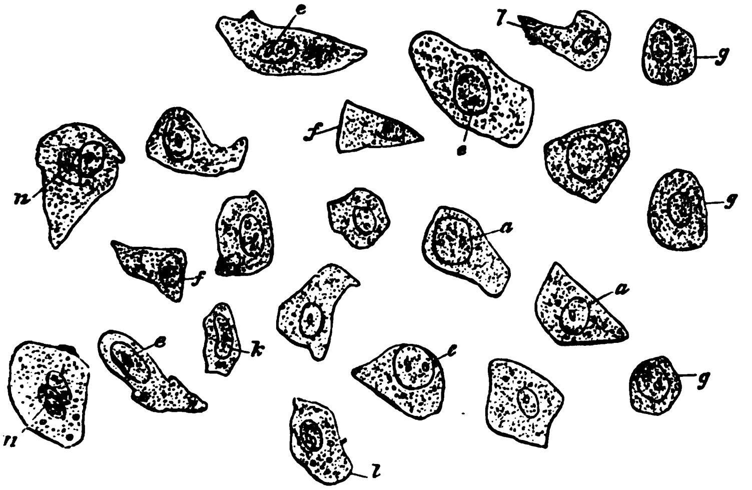
"In regard to the cells themselves of cancer, although we stated their forms as very variable, yet many of them are modifications of the *polygonal*, which may be considered the type. In explanation of the theory of the shape and size of various cell-membranes, we would refer the reader to Professor Schwann's views;[1] undoubtedly, as he supposes, the close crowding together, and the processes of endosmose and exosmose, may be the producing cause. Thus, we observe that in hard firm tumors, particularly those of the breast and ovaries, the cells found are exceedingly irregular, sometimes nearly triangular, Fig. 92, *f*. The ovoid or spherical are more frequently met with in soft or medullary cancer, Fig. 92, *g*, where there is but little pressure, although its juice appears often to be but one mass of cells. It is rare, however, that perfectly round cells are met with, but very generally the angles are well rounded in those which appear to be derived directly from the *polygonal form*, the diameter of which is very variable, ordinarily from $\frac{1}{75}$th to $\frac{1}{25}$th of a millimetre. One peculiarity of this, as of the other forms of cancer-cell, is the presence of the granulations of different sizes in their interior; whereas, in epithelial cells, the interior is generally, when fresh, of course, homogeneous. In cancer, we find the three varieties of granulations given by M. Robin;[2] *first*, the very fine black dots found in all organic elements, and named by the French, very appropriately, *poussière organique; secondly*, the gray granulations, a form somewhat larger; and, lastly, the fat granulations distinguished by the refraction of the light.—This first variety of cells contains nuclei, having in their interior invariably one or more nucleoli, both of which retain the characteristic points described above. The large size of the nucleus, in proportion to the diameter of its cell, will at once strike the eye of the careful observer. The variable position, also, of the nucleus within the inclosure, appears to us to be peculiar to cancer; in cells of other structures, the rule is to find the nucleus very nearly in the centre, except with fibro-plastic cells, where the nuclei appear to have a peculiar affinity

[1] "Microscopical Researches into the Accordance in the Structure and Growth of Animals and Plants," by Th. Schwann. Sydenham Soc. edit.

[2] Tableaux de Anatomie, &c., par Ch. Robin. Paris, 1851.

for the walls. All varieties of cancer-cells contain very frequently two or more nuclei; whereas, the epithelial, more particularly those found

Fig. 92.



Forms of cancer-cells derived from the polygonal or type variety. *g*. Spherical cells. *a*. Dark contour of inclosed nucleus. *e*. The nucleolus. *k*. A nucleus with its contour pressed out of shape. *l*. A form of cell frequently seen, where there is a deficiency of part of the wall. *f*. From pressure rendered triangular.

on the surface of the body (where there is most danger of confusion and doubt), but rarely have more than one. Moreover, the cell of epithelium is much larger than that of cancer, yet the cancer nucleus is twice as large as that of epithelium, as is also the nucleolus, compared with that found in epithelium.

"*Caudulated Cells*.—This variety of cancer element appears to be considered *the* cancer-cell by persons unfamiliar with the microscope. The French pathologists speak of it as *la cellule en raguette*, Fig. 93. Its general aspect is the same as that of the preceding, the only difference being the prolongations, one, two, or three in number, branching off from the body (so to speak) of the cell; sometimes there are as many as five projections. There is no regularity about them, as the reader may perceive in the plate; indeed, they frequently take the most grotesque shapes.

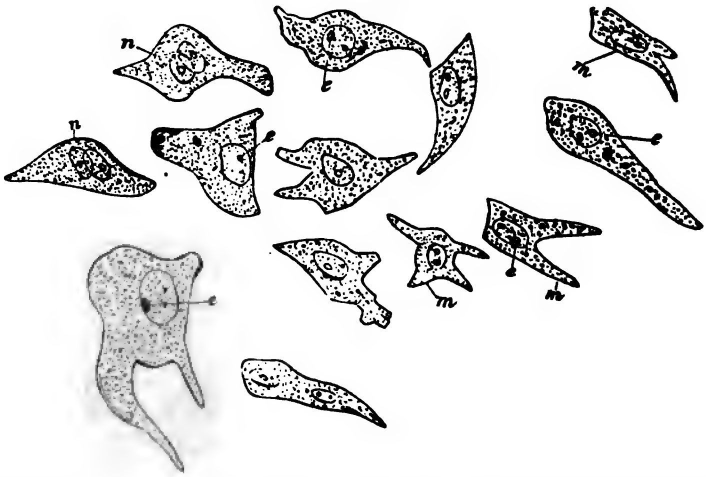
"This form is met with more or less in all cancerous tumors, but invariably in those of the bladder; cancerous degeneration of the parotid often contains them in considerable abundance.

"*Fusiform Cancer-Cells*.—(Fig. 94.) This shape is caused by a swelling in the centre, with the ends pointed, forming often a very acute angle. It is found mixed with the other forms in all parts of the body; but always more numerous in cases where the disease has attacked the bones. M. Robin[1] says that he has never examined cancer of the bones without finding this variety. It is this form which Mr. Bennett confounds

[1] MS. Notes of his Cours d'Histologie, 1850.

with fusiform fibres of fibro-plastic tissue (Fig. 95), making no distinction between them, but describing them together under the name of *fusiform*

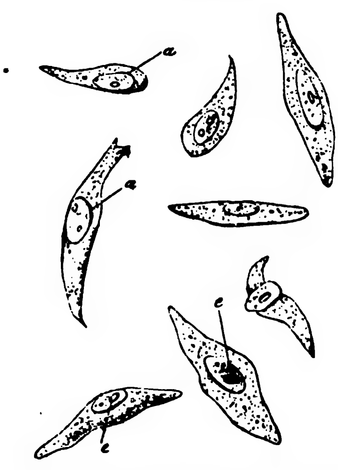
Fig. 93.



Caudated cancer-cells. *m*. The most usual forms. *n*. Cells containing double nuclei; cancer of the bladder invariably contains this variety.

corpuscles. Except some similarity of shape, we cannot see how they could be mistaken for each other. We ask the reader to compare the

Fig. 94.



Fusiform cancer-cells, found in great abundance in cancerous disease of bones. *a*. The nucleus, which, in this variety of cell, is almost constantly ovoid. The transverse diameter of the cell, and the size of the nucleus in proportion to the cell, together with the characteristic nucleolus, distinguish this variety from the fusiform fibro-plastic element.

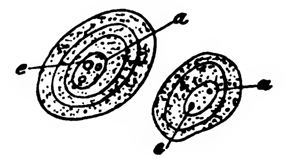
Fig. 95.

Fusiform corpuscles of fibro-plastic tissue. 4. The narrow and long fusiform cell, containing a nucleus (5) with a small dot in its centre for a nucleolus; average length of cell 1-12th millimetre. (Magnified 556 diameters.)

drawing of these two things, and he will at once see that the cancerous is double in width and length; moreover, its nuclei are much larger, and the nucleolus is much smaller in the fibro-plastic, where the absence of the clear bright centre, &c., may be noticed.

The *Concentric Cancer-Cell*, Fig. 96, is formed of an ovoid or spherical body, surrounded by concentric rings, so as to give the peculiar appearance of circles around a centre, increasing in size as they get further out. The centre resembles in every respect the ordinary cancer nucleus, and sometimes other nuclei appear between the circles, and occasionally a nucleus is seen pressing against the outside of the cell wall. It is not known how this variety of cancer constituent is formed, and we forbear giving any of the conjectures in regard to them. Sometimes a mass of epithelial cells are pressed together, and present somewhat this appearance. This cell is met with but rarely, and but few in a specimen; it is more likely to be seen, says M. Robin, in the uterus, breast, and ovaries, than elsewhere; it never forms the basis of the tumor, but is merely accessory. According to Robin, it exists more frequently in the form of cancer tissue, which, in consistence, is between scirrhus and encephaloid.

Fig. 96.

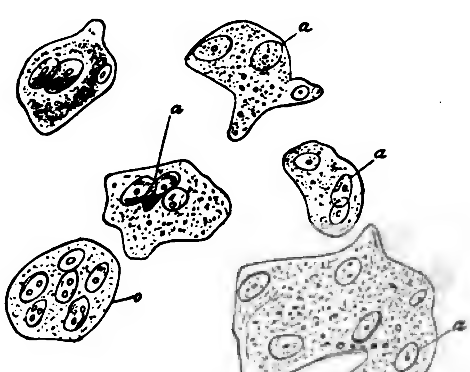


Two concentric cancer-cells. *a.* The cancer nucleus, the size of which is always in proportion to the innermost circle. *e.* The brilliant nucleolus.

"Having ourselves but one drawing of a distinctly marked specimen of this cell, we borrow for our plate one from M. Lebert.[1]

"The *Compound or Mother Cell of Cancer*, Fig. 97, is of very varia-

Fig. 97.



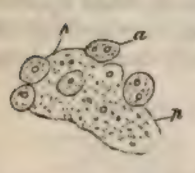
Compound cancer-cells, containing three or more nuclei. *a.* Nucleus; when there are more than one nucleus within a cell they are smaller than the single nucleus. *o.* From Lebert.

ble shape, as the drawings show. They have received this name from the views entertained by some authors, more particularly Kuss and

[1] Physiologie Pathologique. Atlas, Plate XVIII.

Bruch, of their splitting up into smaller segments and multiplying by division. They contain often three, four, or more cancer nuclei. We ourselves have never seen more than seven within one cell, although Lebert gives a drawing of one containing as many as nine. Some consider that secondary cells are formed within the parent one, and are let out by the rupture of the outer wall. It is, however, mere conjecture.—The last form in which these elements are exhibited is where a number of nuclei appear to be glued together, as it were, by the amorphous blastema in which they are generated, without there being any recognizable cell-wall around them. M. Robin[1] calls them *plaques à noyaux multiples*. The size of the envelop about them prevents them from being confounded with anything else. These *agglomerated nuclei* (Fig. 98) are nearly as rarely met with as the concentric cell.

Fig. 98.

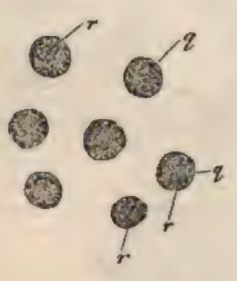


Agglomerated nuclei. *a.* Nucleus. *p.* amorphous uniting tissue.

"All these varieties of cancer element can be seen in the same specimen, although, as we mentioned in speaking of each, they have separately organs of selection. Cancer-cells, of course, like homomorphous elements of the organized animal or plant, have their periods of growth, and development, and decay; their progress to maturity may be sometimes arrested, and account to us in some measure for the great variety of appearance, structure, and size. For some interesting remarks, in regard to the retrograde metamorphosis of all tissues, both normal and pathological, we would refer the reader to an article by Dr. Burnett.[2]

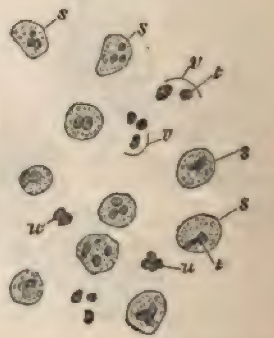
"Out of the body, cancer elements change very rapidly, more so than

Fig. 99.



Pus-corpuscles, magnified 833 diameters. *q.* Type form before the addition of any reactive. *r.* Outline of nucleus seen surrounded by thick granulations.

Fig. 100.



The same after the application of acetic acid. *s.* The irregular contour of the corpuscle freed from the granulations, leaving the nuclei clear. *t.* Characteristic nucleus without any nucleolus. *u.* Free nuclei, the walls having been destroyed. Diameter of pus-corpuscle varies from 1-100th to 1-80th millimetre, that of the nucleus 1-333d. *v.* Remnant of contour.

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[1] Tableaux d'Anatomie, 1851.

[2] American Journal of the Medical Sciences, July, 1851.

any one element we have met with. Often, in the course of the first day, they become degenerated by the appearance of fatty granulations, which often hide their distinctive characters. Unfortunately, they cannot be preserved in any fluid. Alcohol coagulates the albuminous cell-wall. Mixed up with what we have designated cancer elements are often found crystals of cholesterin and of triple phosphates of ammonia and magnesia, filaments, fat-globules, crystals of margarine, pus (Figs. 99 and 100), &c. Wherever there is inflammation, especially of a chronic character, we are apt to find fibro-plastic elements; consequently, we must not hastily conclude, because we find them in a tumor, that there is nothing else there. The importance, therefore, of examining thoroughly, as far as possible, each portion of the specimen, cannot be urged too much. If but one cancer-cell be found, it is conclusive. That which has been designated *melanotic cancer*, is merely a mixture, with true cancer elements, of free pigmentary granulations, or of the peculiar cells of pigment."

"We give, however, that others may compare them, the histological elements with which Mr. Bennett thinks cancer can be confounded.

Fig. 101.

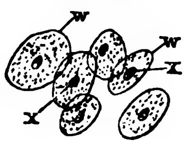


Fig. 102.

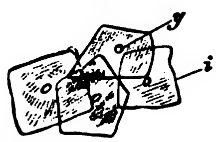


Fig. 103.

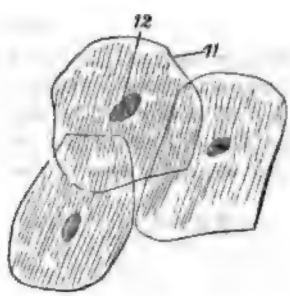


Fig. 101. Young epithelial cells (from Lebert's plate). *w.* Cell-wall filled with few and small granules. *x.* The nucleus, very small in proportion to cell, and containing no nucleolus.

Fig. 102. Tessellated epithelium. *y.* Nucleus without nucleolus, diminutive in proportion to cell. *z.* The cell with homogeneous minute granulations filling up the centre. Diameter of the cell when taken from the skin 1-10th millimetre.

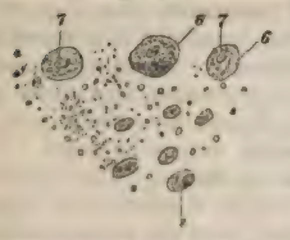
Fig. 103. Buccal epithelial scales, magnified 555 diameters, to show more clearly their dissimilarity to cancer elements. 11. Irregularly polygonal contour. 12. The characteristic nucleus without any appearance of a nucleolus, which is rarely met with in epidermic cells, or in those coming from the buccal surface.

"Fibro-plastic elements possess a peculiar interest in being the only ones where there is any ground for seeing a resemblance to cancer elements. They were first defined by M. Lebert, who thought them always the product of disease. Further researches have convinced him that such is not the case. In the healthy subject, they are found in the bladder, ovaries, liver, mammary gland, uterus, &c. According to Robin,[1] the internal membrane of the Graafian vesicle is the only membrane, in the state of health, which is formed altogether of it."

[1] MS. Notes of his Cours d'Histologie, 1850.

"We give the true fusiform corpuscle (Fig. 95) of this tissue, the length of which is often as much as from $\frac{1}{16}$th to $\frac{1}{31}$st of a millimetre. The narrowness of their width, the smallness of their nuclei, the nucleolus, and, indeed, the whole aspect, would prevent, we should think, any one who is familiar with microscopic investigations, from confounding them with anything else. The fibro-plastic cells and their free nuclei (Fig. 104) could be mistaken for cancer by a superficial observer. They are ovoid, and sometimes polygonal, varying in diameter from $\frac{1}{200}$th to $\frac{1}{66}$th of a millimetre. The appearance, however, of the nucleus itself with the nucleolus, differs very widely from cancer, the granulations in their interior are very much finer, and of more uniform size than those found in cancer. The free nuclei of fibro-plastic tissue are so much smaller as to be easily known when met with."

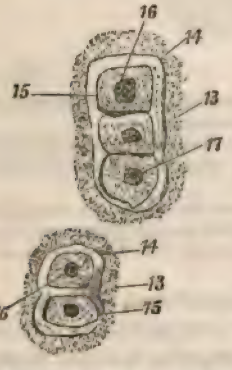
Fig. 104.



Spherical fibro-plastic cells, found in the uterus and in other organs in the healthy subject; also as the result of chronic inflammations; and forming, with the preceding variety, the basis of true sarcomatous tumors. 6. Well-marked cell. 7 and 8. Nuclei inclosed in cells or floating free; transverse diameter 1-200th millimetre.

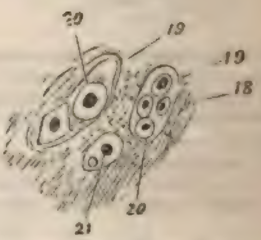
"Mr. Bennett tells us, that when enchondromatous tumors become softened, and the cells escape from the cavities, they resemble very closely cancer. It has never been our good fortune to meet with any such cases; but we confess we cannot understand how, even if the cells were free, they could be taken for those of cancer. Compare them (Figs. 105 and 106) with any or all of the varieties of cancer element, and remark the difference of shape, &c.

Fig. 105.



Cartilage elements taken from the condyles of the femur. 13. Hyaline tissue. 14. Excavated cavity. 15. Cartilage cell. 16. Nucleus. 17. Nucleolus very frequently drowned by the fatty granulations.

Fig. 106.



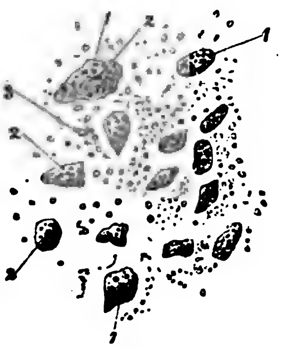
Costal cartilage. 18. Hyaline substance. 19. Cartilage cavity. 20. Cell. 21. Nucleus.

"In Fig. 99, we have given a drawing of *pus*, before the addition of any reactive, and, in Fig. 100, we have given the same corpuscles, acted upon by acetic acid. It will be noticed that with a high power, fre-

quently a dim outline of the nuclei can be seen when the corpuscles are unmixed with any reagent. We are glad to have the support of Bennett and Robin in stating that there is no mucus-corpuscle. What has been so called was either pus, so easily produced on mucous membranes, or epithelial nuclei.

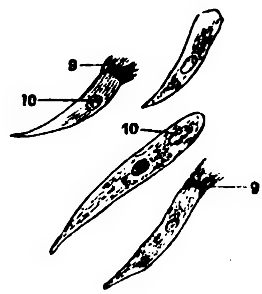
"Thinking it would be not uninteresting to the reader to compare the element of tubercle with that of cancer, we give (Fig. 107) several cor-

Fig. 107.



Corpuscles of tubercle (833 diameters). 1. Corpuscles found in softened tubercular matter; a small, irregularly formed globular body, with neither nucleus nor nucleolus, measuring 1-142d millimetre in diameter. 2. Interior granulations. 3. Free loose granulations.

Fig. 108.



Cylindrical and ciliated epithelial elements, found in the nasal fossæ, trachea, Eustachian tubes, in the intestinal canal below the cardiac orifice. 9. Hair-like extremities, which, during life, are constantly in motion. 10. Nucleus clear in the centre.

puscles found in a specimen of softened tubercular lung handed to us while copying off these remarks; from the first preparations examined we could have given almost any number, but the few we have drawn are perfect type specimens."]

The *origin* of cancer is a subject of the deepest interest, but the first and most important step of the process is entirely concealed from us. M. Simon views a cancer as "substantially a new excretory organ," a growth which arises for the purpose of eliminating from the system an unhealthy matter which is generated within it. Such a function, however, is surely not fulfilled by all instances, even of encephaloid, and still less of the other species. It rather seems that we should recognize in cancer a grave alteration of the normal formative powers; those real, but occult influences which determine that—here bone, and there muscle, and there nerve shall be produced. We know something of the disturbances of nutrition occasioned by unhealthy conditions of the blood *quoad* its chemical composition; we know something of the origin of rheumatism, and gout, and syphilis, and of the effects they produce, but they are very different from the phenomena of new formations. Surely, the arising of a fibrous tumor, an enchondroma, or a cancer, implies a very different kind of action to that which is observed in any blood disease. We certainly believe the blood to be affected, probably in its chemical composition, as well as, and most importantly, in its

vital endowments, but we do not think it is the only seat of primary alteration. Were it so, how would it be possible to account for the constant preference manifested by scirrhus, and by other tumors, for particular sites? We conceive, then, that in the case of cancer, the blood and the general system, but especially some particular part, having suffered some unknown deterioration or perversion of their vital power, a minute quantity of blastema, exuded in the specially weakened part (perhaps in consequence of a blow or other injury), commences to develop cell and fibre structure, which soon constitutes a new growth, endowed with powers of assimilation and vegetation to an almost indefinite extent. The tendency to cancerous disease, that is to say, the deterioration of the blood, and of the assimilative powers, may exist for a long time before it expresses itself in the tangible reality of a tumor; but when this is formed it becomes an engine for multiplication of similar tumors, and intensification of the cancerous diathesis. We have spoken of cancer as resulting from the development of effused blastema, and this, there is good reason to believe, is invariably the case; in the vast majority of instances, the growth manifestly originates in the interstices of textural elements; in some few it has been found in the coats of the veins, but in none has it been certainly proved to have originated in the blood. Cancerous growths have, indeed, been seen within the veins, but this has been the result of perforation of their walls by formations external to them. *Development* proceeds in cancerous blastema just as it would do in healthy; nuclei and fibres seem to arise in the same way. The former are produced, we believe, with great rapidity, not by any slow process of building up by coalescing granules, but by the formation, at once, of a small spherical body, $\frac{1}{36000}$ inch in diameter, which subsequently enlarges, and presents the sharply defined envelop and the clear central cavity with nucleolar spots, which characterize all nuclei. Often, however, the abnormal nature of the formative process displays itself in the irregular forms which the nuclei assume, becoming largish vesicles or granular globules; or, if they have become included in a surrounding cell, enlarging, so as to represent a cell themselves, while secondary nuclei appear in their cavities. The encasement of several nuclei in one cell is not unfrequently observed, and is a wide departure from the ordinary plan of healthy cell-growth. Fibres are formed partly by development from nuclei, which are often seen elongated, partly, and in greater degree, from the blastema, by splitting up and division; they are rarely as perfect as those of healthy tissue. An albuminous fluid, in varying quantity, is diffused through a cancerous mass, and beyond all doubt partakes of the qualities of the solid structure; in it the bloodvessels may be said to lie bathed, and by it, in consequence, the blood traversing the organ must be contaminated. This same fluid is also taken up, in part, by the lymphatics, and thus readily communicates its own cancerousness to the nearest lymphatic glands. The growth of a cancerous tumor will be more rapid, in proportion as its structure is mainly composed of cells, and contains but little fibre, and is also so situated that it has room to expand freely. Encephaloid, in which cell-structure always predominates, is, notoriously, of most rapid growth, but even

its progress is comparatively slow, while it is confined within unyielding walls, as in the globe of the eye. Laennec supposed that all cancers were originally hard, and that in the process of growth they gradually became softer; this is not so; cancers may be quite soft at their commencement; but still, they do generally appear to diminish in consistence as they advance in age. Tumors, however, of the same date may differ very greatly in consistence. Inflammation may affect cancerous tumors; it is commonly excited by their exposure to the air after they have made their way through the covering parts. It powerfully accelerates softening and decay. Suppuration may take place as the result of inflammation, but the pus is an ill-formed sanious product, mingled with detritus. Cancerous formations sometimes mortify spontaneously, sometimes in consequence of inflammation; in the first case a cure has been known to take place, and to attain this artificially is the object of various escharotic applications. It is evident that it is only in instances of cancer, whose powers of vegetation are feeble and sluggish, that such a proceeding can be successful. There are two other changes which cancer occasionally undergoes, and which may result in a cure. One is that called *Saponification*, by Rokitansky, which, however, seems to be simply fatty degeneration. It is this change, in an early stage, which constitutes *Carcinoma reticulare;* it occurs with formation of granule cells, or independently of them. The other change is a shrinking and contraction, a kind of drying up of the cancerous growth with deposition of calcareous matter, analogous to cretification of tubercle.

The term primary is applied to cancerous growths, originating for the first time in the system, secondary to those that are in some way derived by dissemination from the primary. We have already alluded to one mode by which secondary cancers are established, viz: that through the medium of the lymphatics; and we have stated that it is the cancerous blastema which is absorbed, and which gives the impulse to cancerous development in the glands. This, there can be no doubt, is equally capable of propagating the infection as any solid germs to which it is in fact equivalent. If in some instances it does not do so, it is because the natural assimilative power of the gland tissue resists the infectious tendency of the blastema, and perseveres in its own normal mode of nutrition and action. In the same way, we believe that the bloodvessels traversing the cancerous mass readily imbibe through their delicate parietes the diffused blastema, and that the blood thus contaminated, when it arrives in a suitable nidus, deposits there a blastema, which, under favoring circumstances, gives rise to a secondary tumor. The nidus is most often the next capillary plexus at which the blood arrives; thus, cancer of the breast occasions cancerous tumors in the lungs in almost all cases in which it also affects the liver, but it often produces cancer in the lungs without any occurring in the liver; cancer originating in the stomach occasions similar disease in the liver to which its veins proceed, before it produces any in the lungs. It is easy to understand that a suitable nidus is requisite, and that, if the organ first traversed by the blood proceeding from the infected part does not afford such, the material will be effused in vain, the seed will not germinate in a soil unsuited to it. The

recognition of the efficiency of cancerous blastema to produce similar formations is of some importance, as it does away with the difficulty of supposing that solid germs are introduced into the circulation through some breach in the walls of the vessels. These may, doubtless, sometimes make their way into the blood, but there is no proof that they do; and we are certain, on mere physical grounds, that the blastema must. The analogy of pyæmia is in favor of our views; Lebert has shown that the serum of pus produced like effects to the entire fluid, and that the *globules* of pus were not the special cause of the multiple abscesses by being arrested in the capillaries, as they were soon destroyed in the blood. Cancerous blastema, or germs, may be communicated also in another way, viz: by actual contact of the diseased with other parts; cases are mentioned by Dr. Budd, and Mr. Simon, which may be considered to prove this, though there is no improbability whatever in it. Fluid blastema might easily be imbibed from a growth by a soft tissue in frequent contact with it, just as a solution of some salt would be. Germs, that is, nuclei and cells, might also be similarly transferred, if any breach of surface in the growth had occurred. Pathologists are generally agreed in attaching little credit to the results of the experiments which have been made relative to the production of cancer by inoculation, or injection of the cancerous matter. Some few are said to have succeeded, but the great majority have failed; in this there is nothing surprising; we quite agree with Dr. Walshe, that the non-production of cancer by inoculation proves conclusively "the absolute necessity of constitutional predisposition" for the development of the disease. A healthy system will resist and overcome by an assimilative force the cancerous poison, just as it will in the case of a dog that of pus, while a weaker system might be infected by it, as the rabbit is by the injection of pus. What has been called the *metastasis* of cancerous tumors has occasionally been observed; see a case quoted by Dr. Walshe, p. 110, from Recamier; in this, it must not be supposed that the tumor existing in one part is removed molecule by molecule to another, but that either a small pre-existing tumor was excited to rapid growth by the wasting and absorption of the other, or that absorption being induced by some local condition, blastema, or germs, were carried in the circulation to the new nidus, where they commenced to germinate and develop afresh. Were such cases of transfer frequent, they would constitute a considerable objection to the plan of causing absorption of a tumor by pressure upon it. It might be reasonably conceived that the structure of the locality in which cancerous blastema was deposited, would influence the kind of tumor that was therein developed, and such does appear to be the case, at least to some extent. Thus scirrhus is far more common than encephaloid in the female breast, which abounds in fibrous tissue; and encephaloid is most frequent in the liver, which contains little of it, and chiefly consists of cells; the muscular walls of the stomach are commonly also affected by scirrhus, as well as those of the uterus, and both these consist mainly of fibres. On the other hand, there are exceptions in the case of the lungs, and of the meninges which are most often attacked by encephaloid. The species of cancer termed epithelial, seems certainly to be determined by the peculiarity of its site, as it

seems almost invariably to be developed on free cell-bearing surfaces. It is generally true that the greater the proportion of fibre in a tumor, the less is its malignancy; especially intending thereby its tendency to contaminate the system, and to destructive ulceration. On the other hand, the more it abounds in cell-growth, and in fluid blastema, the greater is its malignant capacity. M. Simon says, "in proportion as the blastema has suffered itself to undergo a fibrous transformation, in such measure I cease to recognize that which is distinctively cancerous and malignant." Believing this to be in great part true, we must still remember that the abolition of a truly scirrhous growth may be followed by the development of encephaloid; the inactive fibroid structure is still the expression of the comparatively quiescent constitutional infirmity, which may at any time be roused to its more severe manifestations. There is much probability in the common opinion, that cancer is an hereditary disease. Dr. Walshe acknowledges his belief in the disease having thus originated in some cases which he witnessed; and, indeed, most persons might adduce confirmative testimony. Still, actual statistical proof has not yet been afforded. Cancer is, on the whole, a disease of advancing age; it does, indeed, exist occasionally at every period of life; both scirrhus and encephaloid have been observed in the fœtus, and encephaloid is not uncommonly seen in infants and children of tender years; but the researches of Dr. Walshe show that the mortality from cancer "goes on steadily increasing with each succeeding decade until the eightieth year," so that, taking the mean of both sexes, it attains its maximum between the ages of seventy and eighty. In males the ratio of increase is more uniform than in females; in them, there is a great and rapid increase of mortality between the ages of thirty and fifty, which "lends support to the current belief respecting the connection of the development of uterine and mammary cancer with declining activity and cessation of the genital functions." The influence of sex upon the development of cancer is very striking; Dr. Walshe's tables show that in six years an absolute number of deaths from this disease, in males, was only 3495, compared with 10,146 in females, although the mean rate of mortality in the male exceeds that in the female. A sanguineous temperament is considered, by several who have paid attention to the question, to predispose in some measure to cancer. More powerful are the effects of mental distress, and of the refining, but enervating influences of civilized life; at least, such are the conclusions to which the evidence that can be obtained at present seems to point. A fact of a rather opposite import which Dr. Walshe establishes, is that a town life has no greater influence than a country one, in promoting the development of cancer; in fact, a greater number die of cancer in the country, than in the towns; this applies to the mean of both sexes, but taking the females separately, the mortality from cancer among them is greater in the town than in the country.

An injury to a part (the observation is most common in the case of mammary cancer) seems to be often the exciting cause of cancerous development, and it is probable enough, and accordant with the analogy of growth, that a weakened part should offer the least resistance to the localization of constitutional disease. However, the immunity from can-

cer observed in the Parisian prostitutes, and the result obtained by Andral, with regard to cerebral carcinoma, as following injury, show that but little is to be attributed to this as an exciting cause. Habitual irritation of the stomach by alcoholic liquors has no effect in inducing carcinomatous disease of this organ, though it may have sometimes produced a state of simple induration, which has been mistaken for scirrhus. It is, however, not to be denied, that irritation may in some cases determine the formation of a cancer; as, for instance, in the scrotal cancer of chimney-sweeps, which certainly seems to be called into existence from a latent predisposition by the irritation of soot.

In concluding this subject, we may offer a few remarks with reference to the effects of removing cancerous tumors by operation. In the first place, it is quite clear that the disease is manifestly constitutional, and that no sound, real cure can be expected from merely removing its external development. Secondly, it is matter of experience, that in not a few instances surgical interference with one tumor has provoked the speedy appearance of several others. Thirdly, any attempt at removal is useless; nay, may be absolutely injurious, unless every particle of cancerous structure is taken away. Fourthly, epithelial cancers seem least prone to return after removal; encephaloid invariably does, and mostly with great rapidity; scirrhus may be checked in its progress, but its return can very rarely be prevented. The check which may be given by operation to the progress of cancer depends on the circumstance before stated, that a tumor, once formed, becomes an instrument for the multiplication of similar tumors and intensification of the diathesis. It must require a combination of favorable circumstances, or a great intensity of the diathesis, to insure the development of effused blastema into an heterologous growth; but when this has taken place, then the very growth and vital actions of the structure will constantly generate fresh supplies of cancerous blastema, and thus promote the formation of secondary cancers. The destruction, therefore, of the growth, which thus reacts so evilly upon the system, may be reasonably expected, *if it do not aggravate*, to delay the cause of the disease. But the misfortune is, that, as above stated, it does sometimes aggravate, and that fearfully, a previously indolent cancerous diathesis. Dr. Walshe says, "excision of a tumor seems to awaken a dormant force, cancers spring up in all directions, and enlarge with a power of vegetation almost incredible." Why this should happen, we do not know; but we may conjecture that when the original diathesis is slight, the formation of a tumor may tend in some degree to localize it, and leave the system in a somewhat healthier state, provided the tumor itself be chiefly fibrous, and produce but a small amount of blastema. The removal of the indolent tumor may be analogous to the cure of fistula in ano in a person of phthisical tendency. The two principles referred to of the cancerous tumor, in one case acting as a cause, increasing the force of the disease, and in another retarding it, are not contradictory, though opposite; they will prevail in different degrees in different instances, according to the kind of tumor and other circumstances.

CHAPTER V.

FOREIGN bodies of very various kinds are not uncommonly found in the living organism, having made their way in through the natural orifices, or having been introduced through wounds. They often occasion a great deal of irritation, excite inflammation and suppuration, and in this way become eliminated. At other times, they remain for years without producing any symptoms, and are only discovered after death. When this is the case, they are generally inclosed in a fibroid cyst, which isolates them from the adjacent textures, and prevents them causing irritation. We recently found a bullet thus capsulated in the abdomen of a cat, close to the left kidney; the animal was in perfect health. Dr. Walshe quotes a case in which an ounce of arsenious acid was swallowed without producing any fatal effects; after the lapse of a year the suicidal attempt was repeated, and with success. The arsenious acid of the former attempt was found inclosed in a cyst, which had evidently prevented it from exerting its poisonous influence.

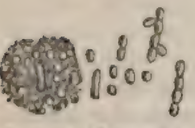
PARASITES.

These are organized formations existing in a living organism, but not connected with it by any continuity of tissue, and possessed of an independent life. In these respects they differ from the class of new formations. They have been often supposed to be produced in a spontaneous manner, originating as by a kind of necessity from morbid products; but the progress of inquiry has proved, almost completely, that this is not the case; that they are developed from sporules or ova, just as beings of a higher rank are; and that the cause of their appearing in certain localities, under certain morbid conditions, is, that in such cases the germs find a suitable soil or nidus wherein they can be developed. We believe that recent researches have also rendered it very probable, if not certain, that the same species of parasite may develop itself very differently in different localities; so that some, which were formerly regarded as distinct species, are now considered to be only varieties of the same.

Vegetable Parasites. These are microscopic growths belonging to the lowest class of the vegetable kingdom. They are found most commonly on diseased mucous surfaces, sometimes in exudations, but it cannot always be clearly determined whether they are the cause of the diseased state, or whether (as is more probable) the diseased tissue has

merely afforded a suitable nidus for their development. We enumerate those which have been observed, after Rokitansky, as follows: (1.) The mycoderm of favus. It consists of minute, transparent, round or oval vesicles, often united in branching threads. This arrangement results from their mode of growth by elongation and division of the cells. M. Lebert has observed a rotary movement of the separate or united cells similar to that which has been detected in other cryptogamia. The vegetable growth is inclosed in a kind of capsule, which is marked on the surface by a funnel-shaped depression, and is of a sub-conical shape beneath, where it is implanted in the skin. Experiments to reproduce the disease by inoculation with the mycodermic sporules have scarcely ever succeeded; it seems therefore probable that they do not constitute the essence of the disease. (2.) A similar fungous growth has been found in the sheath surrounding the root of the hair in sycosis. (3.) In porrigo decalvans, a growth of very minute fungi has been detected within the roots of the hairs. (4.) A similar one has been found in the hair roots in plica polonica. (5.) Also, in pityriasis versicolor. Croupous, aphthous, and diphtheritic exudations upon the mucous surface of the mouth, pharynx, œsophagus, intestine, and larynx, are often found to contain minute fungi very similar to those of favus, except that they are longer and more slender, and more distended by reproductive granules at their terminal extremities. They have also been observed in gangræna oris, in sputa from a patient affected with pneumothorax, in the sordes of the teeth, &c.

Fig. 109.



Drawing of Mycoderm of Favus — partly from Lebert.

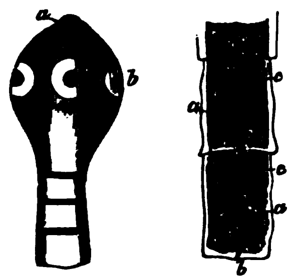
Animal Parasites. Infusoria (vibriones and vorticellæ) are not unfrequently found in pus, and probably in other fluids. Dr. Bence Jones has discovered vibriones in the urine of a child the moment after it was passed. The following *insects* are known as human parasites: the common flea (pulex irritans), the chigoe of the West Indies (pulex penetrans); the pediculus capitis; the p. pubis; p. vestimenti; p. tabescentium; the common bug (cimex lectularius). To these may be added the harvest bug, which, like the chigoe, burrows into the skin probably for the purpose of laying her eggs. Among *arachnida*, we find two species of *acari*, that frequent, the one the epidermis, and the other the sebaceous follicles. The acarus scabiei (sarcoptes hominis) is a minute whitish creature, about $\frac{1}{100}$—$\frac{1}{80}$ in. in size; it has no true head, but is provided anteriorly with proboscis-like mandibular organs furnished with four bristles. There are eight legs, "four anterior are inserted into the thorax by the side of the proboscis, are jointed, and furnished with hairs and bristles, the last joint of each terminating in an adherent disk. The posterior legs, without adhering disks, terminate in very long bristles." (Vogel.) The animal burrows in the epidermis, and forms minute channels, at the end of which it may often be discovered; it does not inhabit the vesicles or pustules which constitute the eruption, and are simply excited by the irritative proceedings of the acarus. The acarus folliculorum is proportionally much longer, about $\frac{1}{125}$—$\frac{1}{75}$ in. long. $\frac{1}{500}$ in. broad; its head has two lateral palpæ with an inter-

vening proboscis; its thorax is supported by four pairs of very short legs with terminal claws; its long abdomen gradually diminishes to the end, and contains some granular matter and oil-vesicles. It inhabits the hair follicles and sebaceous glands in any region of the skin, where it may be found in the healthiest persons. It does not appear to excite any disease. The following *Helminthic* parasites infect man: (1.) *Filaria medinensis*, the Guinea worm, from six inches to twelve feet long, about as thick as a piece of packthread. It makes its way into the subcutaneous tissue of the lower limbs and some other parts, where it remains a variable time without exciting any particular symptoms; but when its progeny are approaching the period for their extrusion, it makes its way out, or is extracted by hand. The symptoms of this period are sometimes slight, sometimes very severe. If ruptured during extraction, the young escape into the cellular texture and excite an unhealthy suppuration. (2.) *Filaria oculi humani*, discovered in the surrounding fluid and in the crystalline lens. (3.) *Filaria bronchialis*, once found in diseased bronchial glands. Other filaria have been found in the blood and in the urine (spiroptera hominis, dactylius aculeatus). (4.) *Trichina spiralis* is a minute round worm, $\frac{1}{24}$ to $\frac{1}{36}$ inch long, with an intestinal canal and distinct oral and anal openings. It occurs inclosed in a transparent cyst, which is situate in the interior of the fibres of voluntary muscle, separated by the sarcolemma from all surrounding textures. Sometimes there are two or three worms in one cyst. Sometimes the cyst contains calcareous matter, in which case the worm is most commonly dead. The pathological significance of this worm is not very apparent; it has often been found in persons who have died of different diseases, and even in those who perished while in health and vigor. (5.) *Tricocephalus dispar*, a thin filiform worm, one and a half to two inches in length. Its anterior two-thirds are quite capillary, and pass rather suddenly into the thicker posterior portion. The sexes are distinct; the male has a long penis invested with a proper sheath. The female produces numerous ova, few of which are probably developed. The worm is found chiefly in the cœcum, adhering by its head to the mucous membrane; it is sometimes solitary, sometimes occurs in great numbers; it does not appear to produce any remarkable symptoms. (6.) *Ascaris lumbricoides*, a round worm, pointed at both ends, from six to fifteen inches long, of a grayish and sometimes red color, sufficiently translucent to allow the viscera to be seen, marked by two lateral lines corresponding to the principal vessels, and by two others less distinct, and corresponding to the nervous cords on the dorsal and ventral surface. The head is separated from the body by a slight constriction; at its extremity is the mouth, surrounded by three tubercles. The anus is situated on the under surface, near the extremity of the tail. The reproductive organs consist, in the male, of a single seminal tube, three feet long, terminating in a reservoir about an inch in length, which communicates with the base of the penis: in the female, they comprise the vulva, situated at the junction of the anterior and middle thirds of the body; a vagina, five or six lines long; a uterus, which divides into two long, tortuous oviducts, gradually diminishing to capillary ovarian tubes, which are conspicuous

by their whiteness, as they are coiled around the intestinal canal. They inhabit chiefly the small intestines, but roam about occasionally up into the gall-ducts, the stomach, the œsophagus, and have even made their way into the nostrils and into the mouth, as happened in a girl under our care. It seems at least doubtful whether they ever perforate the coats of the intestine and get into the peritoneum. Sometimes they are very numerous; one patient passed as many as four hundred and sixty in a fortnight, but this is not common; however, one would rather be inclined to expect, from the appearance of one, that there were others remaining behind. They have been known to accumulate so as to obstruct the intestines; more commonly, they produce only some amount of irritation, or even no symptoms at all. We think we have seen a case of convulsive paroxysms depending on the presence of these parasites in the bowels. (7.) *Ascaris vermicularis;* a minute, white, thread-like worm, of separate sexes; the male about one and a half lines in length, the female five or six; the former has a spirally coiled tail, the latter a straight, tapering, and very delicate one. There is a transparent tuberosity on the head, with a kind of alar membrane on each side. They inhabit the rectum in vast numbers, and may crawl out and get, in females, into the orifices of the adjacent canals. They occasion very distressing irritation, perhaps in consequence of their restless disposition, which has obtained for them their name of ascarides (ασκαριζειν, to leap). (8.) *Strongylas gigas*, a formidable large round worm, which attains sometimes three feet in length, and is of a blood-red color. The male, as usual, is the smaller, is marked by circular striæ and "shallow longitudinal furrows;" at its posterior extremity it has a funnel-shaped pouch, from which a slender penis protrudes. The female has a straighter and more obtuse tail, and at one or two inches' distance from it the vulva. It inhabits the kidneys, and causes more or less destruction of this organ. Among the *trematoda*, we are acquainted with three species of distoma, which have been found, though rarely, in the human subject. The *d. hepaticum* and *d. lanceolatum* are both flat, lancet-shaped worms, of a yellow-white color, with two suckers, one of which situated at the head forms the mouth; the other is on the abdomen, and is imperforate. The orifice of the sexual organs lies between these two. They are hermaphrodite. The d. hepaticum is the larger, being eight to fourteen lines in length, and from one and three quarters to six lines in breadth; its intestinal canal is ramified. The d. lanceolatum is only two to four lines long and one broad; its intestine is bifurcated. They have been found in the gall-bladder and ducts, and in the v. portæ and its branches. *Distoma oculi humani* is of minute size, and has been found in the fluid surrounding a cataractous lens. *Cestoidea.*—Two very common parasites of the human subject belong to this order, the tænia solium and the tænia lata. They are both flat-jointed worms, of a whitish-gray color, and attain, very often, an immense length. Their joints, laden with ova, frequently separate, and are discharged from the system, while fresh ones are formed in succession behind the head. The one do not appear to be developed in the organism inhabited by the parent worm. The head of the tænia solium is at the anterior smallest part of the worm; it is provided with four lateral suckers, between

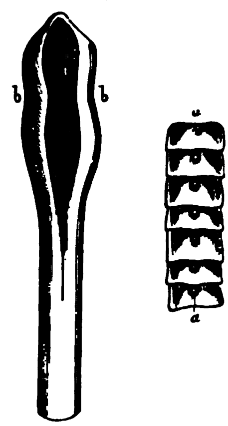
which is a circle, sometimes supporting a double row of hooks; in the centre of this circle there is a minute opening, that of the mouth. "The anterior segments," Professor Owen says, "are feebly represented by transverse rugæ; the succeeding ones are subquadrate, and as broad as long." Advancing posteriorly, the segments still increase in length and size; the anterior part of each is overlapped by the broader posterior

Fig. 110.



Tænia solium. Head and joints.

Fig. 111.



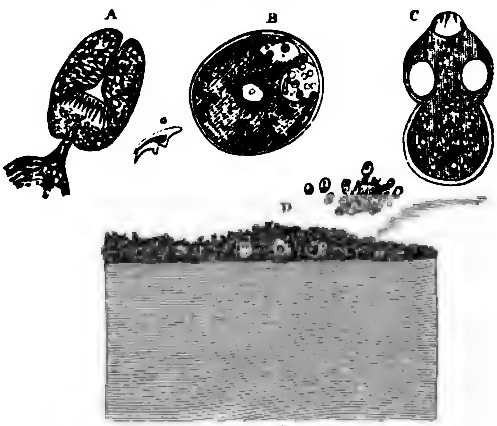
Tænia lata. *a*. Marks the generative orifices in both. From Owen's Lectures.

part of the preceding. At about the middle of the margins of the joints is situate the orifice of the generative canals, which is in successive joints on alternate sides; this constitutes a distinction between the tænia solium and the tænia lata, in which the genital orifices are in the middle of the ventral surface of the segments. The alimentary canal in both tæniæ seems to commence as a single minute tube, which soon bifurcates and forms two divisions, which run throughout the length of the worm, at a little distance from the margins. The generative apparatus is very highly developed in each joint, consisting of a large branched ovarium and a seminal tube, which terminates as a small rudimental penis, situated just anterior to the opening of the ovarian canal. The head of the tænia lata, or *Bothriocephalus latus*, forms an elongated, sub-compressed enlargement, with an anterior obtuse prominence, perforated by the mouth, and having two lateral sub-transparent parts separated by an opaque tract; these have been regarded as depressions (βοθρια), whence the generic name. There is no trace of joints within 2½ inches of the head; these are, at first, feebly marked, then the segments expand posteriorly, and slightly overlap the succeeding ones; their length nearly equals their breadth. The generative apparatus repeated in each segment, as in the tænia solium, is still more developed, and occupies, especially in the hinder segments, by far the greatest part of the space. It consists of complicated tubular ovaries, and a convoluted uterus, on the one hand; and of a large glandular testis, vas deferens, seminal receptacle, and penis, on the other. Certain glands, which seem to furnish a matter

to protect and cement together the ova, open into the uterus, near its termination at the vulva. The multiplication of the joints seems to take place by division of the first, that immediately succeeding to the head, the two parts subsequently enlarging. The substance of the joints in the tæniæ consists of very minute nucleated cells, which doubtless obtain nutrition by absorption of chyle through the delicate integument. There are two layers of muscular fibres, a transverse and longitudinal. The ova are most matured in the posterior segments, which are, of course, the oldest. It is extremely remarkable, and a most merciful provision, that the ova do not undergo development in the intestine which lodges the parent; otherwise, their enormous multitude would speedily exhaust the individual infested by them. The further progress of the ova has not been determined, but it is most probable that after extrusion they (that is, such as do not become abortive) light upon some suitable nidus, where they attain a certain stage of development before they are again transferred to the intestine of some hospitable recipient. The tænia solium is a native of Britain, Holland, Germany, Egypt, and the Levant; the tænia lata, of Russia, Poland, Switzerland, and Eastern Prussia, and of Middle and Southern France: it is clear that it is some peculiarity in the localities, and not in the human inhabitants, which determines this limitation, as a visitor to a foreign country may be attacked by a different parasite to that which is indigenous in his own land. *Cystica.*—There seems to be good reason to doubt whether the animals contained in this order should be separated from the preceding; the formation of the head in both is identical, or nearly so, and the different shape of the body may very possibly depend on its being placed under different conditions of development. *Cysticercus cellulosa* has a conical, glistening, white, transversely wrinkled body, and a caudate vesicle, which assumes various shapes, according to the pressure of surrounding parts. Its size is about that of a pea or bean, in most cases, but when situate in open cavities, as the cerebral ventricles, it may attain the size of a hazel-nut. The body and head can be drawn into the vesicle, and when there may escape notice. The head is somewhat larger than the neck, of a quadrangular shape, with a round sucker at each angle, and provided in the middle, at the base of a conical proboscis, with a double circle of hooks, amounting in number to about thirty-two. In structure, the body and globose tail consist of a well-marked homogeneous membrane, which is covered over internally with a layer of highly-refracting celloid particles. These are very closely packed together in the body, but are less numerous in the neck, and disappear by the caudal appendage; they have very much the aspect of fat-cells, but are shown, by the action of acid, to contain carbonate of lime. They lie upon a stratum of dark, finely-divided molecular matter, which is also spread over the surface of the interior of the caudal pouch. A neutral, slightly albuminous fluid distends this cavity moderately. When the cysticercus lies in the substance of an organ, it determines the formation of a kind of fibrous cyst around itself; this is wanting when it lies in a natural cavity, as the ventricles of the brain. After the death of the animal the body collapses, and degenerates, more or less completely, while calcareous deposition at the same time takes

place, so that there remains at last only a chalky mass surrounded by a fibrous cyst. This parasite occurs in the brain, and in the voluntary muscles; it is sometimes solitary, sometimes in great numbers. *Echinococcus hominis* is a minute, but very interesting and not uncommon parasite, which inhabits, in great numbers, the interior of those large globular sacs which are called acephalocysts or hydatids. One of these consists of an external enveloping cyst, composed of condensed fibrous tissue closely in contact with the tissue of the part in which it lies, traversed by some vessels, and lined on its interior, according to Vogel, by an epithelium. Within this is a second cyst of nearly milk-white aspect, nearly brittle, and yet, elastic in some degree, without any organic connection whatever with the inclosing cyst, and filled, itself, with a pellucid serous fluid, in which are often numerous secondary cysts, either free and floating, or attached to the wall of the parent. These secondary cysts sometimes contain a tertiary, and the tertiary again a fourth generation. The younger cysts are tensely filled with a fluid like that of the parent, which spurts out on an incision being made, while the membrane curls outwards. They are sometimes so numerous as to

Fig. 112.



A. Echinococcus, the head retracted.
B. Echinococcus, the head and cornet of hooks facing the observer.
C. Echinococcus, the head extruded.
D. Section of wall of acephalocyst, with blastematous layer, in which are seen several Echinococci.

occupy great part of the parental cavity; they vary, also, much in size, from that of a small seed to that of a goose's egg, or even more. Their membrane is precisely similar to that of the parent; it is beautifully seen, when a thin vertical section is placed in the field of the microscope, to consist of a very great number of laminæ, which are arranged concentrically, and vary in thickness, from $\frac{1}{5000}$—$\frac{1}{2000}$ inch, according to a recent observation which we made. The substance of the membrane has the chemical properties of coagulated albumen, or fibrin, and there can be no doubt that it is the result of the effusion of liquor sanguinis from the external cyst, which solidifies as it is effused, layer by layer;

and thus, somewhat after the fashion of an aneurismal sac, is formed an interior laminated cyst. But little is known as to the mode of formation of the secondary cysts; what we have ourselves observed amounts to this: On the interior of a primary cyst, a number of whitish opaque spots, of the size of a pin's head, were seen; these were produced by the existence of a number of circularly disposed laminæ, identical with those of the membrane itself, and apparently inclosing a cavity. A little more elevation and isolation of these spots would have produced a secondary cyst; not indeed detached, but sessile, as they often are found to be. The transparent fluid contents of the cysts have a specific gravity of 1.008—1.013, are neutral, or slightly alkaline, contain very little albumen, if any, some extractive and fatty matter, and some salts, especially chloride of sodium. With respect to the echinococci, whose habitation we have now described, they seem to form in a stratum which lies on the inner surface of the parent cyst. This is of opaque oily aspect, and consists of imperfect celloid particles, irregularly shaped, non-nucleated, a little larger than pus-corpuscles, and with faint granular contents, lying imbedded in an oily and granular matter. In this kind of blastema there lie numerous echinococci, which, as in the instance referred to, may be separated, or aggregated together in groups. The groups are sometimes inclosed in a spherical sac, attached by a pedicle to the wall, or the individuals of the group are connected together only by a short branching pedicle, which sends a division to each. The echinococci are about $\frac{1}{235}$ in. — $\frac{1}{78}$ in. long, and $\frac{1}{360}$ — $\frac{1}{100}$ in. wide; they have a head much like that of a tænia, provided with four lateral suckers, and a double row of hooklets, situate at the extremity, around an orifice. The head is separated by a contraction from a thicker roundish body, at the posterior extremity of which is a transverse depression, into which a short delicate pedicle is inserted. This pedicle is the same before mentioned by which the animal is attached to the cyst membrane. The echinococcus is most commonly seen with its head inverted, and, as it were, doubled inwards, so that the animal appears of an oval shape, with an orifice at the part opposite to the attachment of the pedicle, and a narrow canal leading from thence downward to the middle of the interior, where the circlet of hooks is now seen marking the position of the extremity of the head. It consists of a strong, homogeneous membrane, which is covered over, internally, by a soft granulous substance, in which are imbedded several refracting, probably calcareous, celloid corpuscles. There is no appearance of reproductive organs in the echinococci; the young seem to be produced in the blastematous layer we have already noticed. They are described by Mr. Erasmus Wilson as originating in certain transparent cells, some of which are, however, more opaque. Both contain smaller cells, but these are more defined in the latter, and are mostly nucleated. The inclosing membrane of the primary cell ruptures, and the included cells are diffused "in the form of a small opaque patch in the substance of the internal membrane of the acephalocyst." The cells multiply, and form a small globular prominence on the surface of the membrane, while one larger and more transparent cell is to be seen in the interior of the mass. As the germinal patch increases in size, other protrusions

take place around the base of the original one, and develop in the same way. The formation of the echinococcus from the mass of cells seems to take place by their coalescence, and retiring from the centre so as to leave a tubular canal, which Mr. Wilson believes to be modified, so as to form in the body the retractor muscle of the head, and exterior to it the peduncle.

A ciliary movement has been seen by M. Lebert in the interior of echinococci; it does not, however, appear to be a constant phenomenon. We have never seen it ourselves. Among the multitudes of living echinococci, some are found which are shrivelled, altered in shape, more opaque than the others, and filled with an opaque granular mass. These are doubtless dead, and, at the same time, traces of others, which have completely decayed, are discernible in the shape of detached hooklets. When, as occasionally happens, a cyst, such as we have above described, is entirely barren of echinococci, it is properly termed an acephalocyst. This is much more frequently, we think, the case with the secondary and tertiary than with the primary cyst. We cannot hesitate for a moment to regard the cyst productions as simply results of a peculiar exudative process, and not as in the least partaking of the character of animals. The exterior envelop is derived from the natural tissues of the affected part; the interior, which can alone be considered as proper to the acephalocyst, can scarcely be said to be organized, not, we think, certainly so much as the successive layers of false membrane, which are formed upon an inflamed serous surface. Neither can we consider the acephalocyst as a "gigantic organic cell," as all we know of its origin is entirely opposed to such an idea. Its relation to the echinococci is, in all probability, that of affording a suitable nidus for their formation and development; but it is by no means clear why, in some acephalocysts, the animal cells are wanting; or why, in others inhabited by them, no secondary cysts are produced. Perhaps it may be found that the development of echinococci and secondary cysts takes place in an inverse ratio to each other. The cysts, both primary and secondary, are subject to degenerate; they become less tensely filled, their walls softer and more gelatinous, their contents turbid with diffused granulous matter and debris of echinococci. At last they shrivel up into a caseous mass, which is often the seat of calcareous deposit. A variable number of its progeny may decay in this manner, without the parent cyst itself being similarly affected. Inflammation of the external enveloping cyst is the chief cause of the destruction of the primary formation in contact with it; this is sometimes very acute, and leads to the formation of an abscess, which subsequently opens externally, or into some of the adjacent cavities or canals. The debris of the acephalocyst and its contents may be evacuated in this way, and as the cavity contracts and closes a cure is effected. When the inflammation is more chronic, the exudation which is poured out into the cavity of the primary cyst seems to derange the nutrition of the included ones, so that they shrink up and degenerate into mere laminæ, of cheesy consistence; these, together with an oily calcareous residue of the fluid contents, which are gradually absorbed, remain in the interior of the primary cyst, which itself wastes and shrinks like its included progeny. In this way, another kind of cure of

the disease takes place, as the degenerated mass may remain for an indefinite time in the substance of a part, without giving rise to any symptoms. Such degenerated acephalocysts have, it would appear, been mistaken for tubercle. The following organs are occasionally the seat of acephalocysts. The liver, κατ' ἐξοχην, the peritoneum, and the underlying areolar tissue, the muscles, the brain, the spleen, the kidneys, the lungs, the bones. They not uncommonly occur in several parts at the same time. The only injurious effects ordinarily caused by acephalocysts, are such as result from pressure on important parts. It may, perhaps, happen that they cause exhaustion when very numerous.

It may be well to add a caution, with respect to Pseudo-parasites, as they are termed. These are either real animals, which are mingled with the excretions by impostors, or substances which are really produced in the body, but are not animals at all. It may sometimes require a more than ordinary acquaintance with parasitic animals to arrive at the truth.

THE PATHOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.

CHAPTER VI.

GENERAL OBSERVATIONS.

In no organ or system of the human body is there such an apparent want of accordance and definite relation between the symptoms of disease and the structural derangements and changes produced by it, as in the nervous system at large. This is due to various causes. In the first place, our physiological knowledge of the laws governing the action of the nervous system is not on a par with our acquaintance with the processes operating in other organs; chemistry, physics, mechanics, can be called to aid in the investigation of the healthy and diseased states of the bones, the soft tissues investing them, the thoracic or abdominal viscera; and the structure of these parts is more analogous to what meets the eye of the scientific observer in other departments of nature. The only agent to which nervous functions can be compared, eludes our senses, except in its manifestations, as much as the operations of the cerebro-spinal energies themselves; we can judge of galvanism and electricity by the effects they produce, but we do not see their *modus operandi;* and we have not even succeeded in demonstrating, as yet, the existence, in the brain or spinal cord, of a structure which may legitimately be concluded to generate a force, in a manner similar to the production of that powerful agent, which is now metamorphosing the human race. It is, then, manifestly impossible, if we are imperfectly acquainted with the laws governing the nervous system in health, that we should be able, sufficiently, to define and appreciate the deviations from the healthy standard; and, in many instances, we must be satisfied to refer for explanation of the symptoms we meet with, to hypothetical analogies, or confess our entire incapability of accounting for the phenomena before us. A correct appreciation of the symptoms of disease is a point which next may be fairly demanded of us, if we attempt to explain or seek for morbid phenomena after death; thanks to Sir Charles Bell, Flourens, Marshall Hall, Romberg, and other distinguished inquirers of the present age, much has been cleared up, which previously appeared an impenetrable mystery; but our means of physical diagnosis are still but scanty; we are left, in a large num-

ber of cases, to form our opinions from the subjective statements of the patient; and where these fail us, as in early childhood, or in certain forms of disease, the greatest empirical experience may be inadequate to offer an explanation of the symptoms, or even to determine their exact relation to cerebral lesions. The peculiar connection of the brain with our mental powers and the soul, will, necessarily, ever place it in a different position, in regard to physical research, than other organs of the body, and in no department of natural science is the caution more necessary, that we should not mistake that which is unattainable to science, and that which the Creator has allowed to be within our reach; and, valuable as are vivisections, and other experiments, made upon the lower animals, in reference to neurology, we must never forget, on the one hand, the disturbance to the ordinary laws of action likely to be produced by the injuries themselves; and on the other, the chasm which intervenes between the brute creation and the human species. We cannot allude to the subject of the diagnosis of nervous disease, without dwelling, forcibly, on the necessity for availing ourselves of those means which, limited as they may be, are yet not used as uniformly and perseveringly as they are applied for the investigation of other morbid states of the body. A close examination by the eye and by the hand should be made of the cranium, the spinal column, and the courses of affected nerves; the thermometer and the dynamometer should be brought into more frequent use, and more accurate tests of the sensibility and mobility, and other functions of the nervous system, ought to be employed than have hitherto generally prevailed.

The researches of Bright, Frerichs, and others, have demonstrated the close relation of the state of the blood to cerebral disease; and science has shown, what, previously, was purely hypothetical, that the most fatal conditions may be thus induced without any palpable changes being wrought in the cerebral tissues. It does not, however, follow that, because we see no changes, none have taken place. The poison that we know to be in the blood may elude our chemical tests, and yet cause death. Then, seeing how limited our knowledge of the nervous system is, it is not to be wondered at that, although the manifestation of altered function is so great as to force the belief in its altered constitution, it is not in our power to prove the latter to the perception; but, as Dr. Watson remarks, "whatever may be the nature of the unknown, and, perhaps, fugitive physical conditions of the nervous centres, thus capable of disturbing, or abolishing their functions, it is useful to keep in our minds a distinct and clear conception of the fact, that there must be some such physical conditions."

In examining the pathology of the nervous system, we shall adopt the succession usually followed by medical writers, and consider, first, the brain and its membranes; next, the spinal cord, with its membranes; third, the cerebro-spinal nerves, and the subject will be concluded by a summary of what is known with regard to the sympathetic.

CHAPTER VII.

THE DURA MATER.

THE intimate relation existing between the dura mater and the cranium renders it peculiarly prone to sympathetic affections propagated from the bone, and from its proximity to the latter it is most likely to be involved in injuries of a traumatic nature. As on its external surface the close contact with the cranium favors a communication of disease from without, so the relation of its internal surface to the arachnoid induces a liability to communication of morbid action from within. The amount of idiopathic disease discoverable in the dura mater in the dead-house is not great; though we are justified, by its character as a fibro-serous membrane, in assuming that it is frequently affected during life; thus, the headaches complained of by patients affected with chronic rheumatism may often fairly be set down to a diseased condition of the dura mater, both from the resemblance which its symptoms offer to those occurring in rheumatism of other fibro-serous membranes, and from the success resulting from an anti-rheumatic treatment. It is not the general character of rheumatism to cause marked or extensive disorganization when attacking membranous expansions, and there are no especial reasons to expect a deviation from this rule in regard to the fibrous covering of the brain. A considerable difference exists in the adhesion between the dura mater and the cranium at different periods of life, independently of disease; the connection is lax in childhood, and is rendered more intimate and firm with advancing age, as the sutures become ossified and the bones lose their resiliency. It is especially in the latter period that external injuries are liable to implicate the dura mater as well as the bone, and one of the most common effects of blows or concussions is a forcible separation of the membrane, with hemorrhage between it and the bone. The clot may, as elsewhere, be partially or entirely absorbed, and we accordingly meet with it in the various stages of metamorphosis. One form of hemorrhage in this region is connate, and is termed cephalhæmatoma;[1] it is produced by the pressure exerted during parturition, and is generally found in the form of a tumor, varying in size from a walnut to a child's head, on the parietal bone, presenting during labor. As this, however, is rather an affection of the pericranium, we shall revert to it when speaking of the morbid anatomy of the bones. We may state that nature adopts the same process of limitation and absorption in the case of the external as in the internal hemorrhage.

[1] Deriv. κεφαλη, head; and αἱμάτωμα, sanguineous tumor.

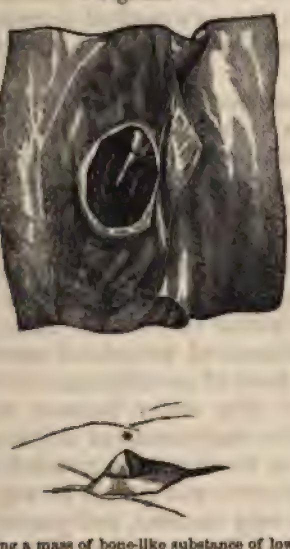
Inflammation of the dura mater presents the features of inflammatory action in fibrous tissues generally; it is never of a very active character, and in many instances of contiguous inflammation, the membrane seems to act as a barrier, to intercept its progress. In the first stage the membrane presents a pinky hue, which is irregularly diffused, and evidently has but little tendency to spread. The injection causes the dura mater to assume a more lax and pulpy condition, and it is more readily detached from the adjacent parts. A stage of infiltration and suppuration, or of effusion of lymph may follow; the latter is the more frequent result of idiopathic inflammation of the dura mater, and may give rise to induration, firm adhesion, thickening, or to new formations, such as the production of bone. In fibrous tissues generally the tendency to lymphatic effusion is rather on the surface, causing an attachment to adjacent parts, whether bone or muscle; in the dura mater, however, interstitial effusion is more frequently met with, on which account the latter is more likely to show the traces of inflammatory action than aponeurotic or fascial expansions. When apparent adhesions occur between the dura mater and the arachnoid, which are not unfrequent, they are rather the effect of inflammatory action and deposition of lymph on the latter than on the former. In such a case the removal of the dura mater cannot be effected without laceration of the arachnoid or the subjacent gray matter, or connecting bands or shreds are found to be drawn out as the membranes are separated. The reparation of solutions of continuity in the dura mater is not effected with the same rapidity and vigor that we see in more vascular tissues. From its contiguity to the skull it is very liable to be implicated in external injuries, which may assume the character of the injury to the bone, being cut, punctured, or bruised; or, we occasionally meet with laceration of the dura mater from concussion at a distance from the point at which the force was applied. The same causes that give rise to hemorrhage between the periosteum and bone elsewhere, frequently induce extravasation between the dura mater and the inner table of the skull; but they are all of a mechanical nature. A form of inflammation that we very commonly meet with in the dura mater is that resulting from inflammation occurring in the internal auditory passages and the cells of the mastoid process. This form is not only secondary, but also of a dyscrasic character; it is met with chiefly in childhood and in scrofulous individuals. As infants may be affected with otitis, it often becomes a matter of difficulty to form a correct diagnosis at the commencement of the disease, when remedies are most likely to arrest it and prove beneficial. The disease may be initiated in the mucous membrane or the bone, and as the morbid process extends towards the cavity of the cranium, effusion of lymph or pus takes place under the dura mater, and the brain itself generally becomes involved. The dura mater is often found black and sloughy, especially over the diseased portion of bone; and it is a curious fact, illustrating the propagation of disease from one tissue to another without actual contact of the morbid process, that there need not necessarily be a perforation of the pars petrosa to induce the inflammation of the dura mater. Mr. Toynbee has found that disease communicated to the encephalon in this manner more frequently induces morbid action in the cerebellum

than in the cerebrum. The same affection, purulent otorrhœa, when of long standing, is liable to induce inflammation in the sinuses of the dura mater; though other injuries, accompanied by purulent discharges, may also give rise to it. This particular form of phlebitis is mainly, if not exclusively met with in early life; when the sinuses become inflamed in manhood, it is the result of external injury. The dura mater in such a case is found more closely adherent to the skull in the line of the inflamed sinuses and their vicinity than elsewhere, and the contents of the sinuses are fibrinous coagula, while their lining membrane is thickened and deprived of its lustre and smoothness, as in other cases of phlebitis. More or less adhesion is generally found to exist between the dura mater and the visceral plate of the arachnoid, when the former has suffered from inflammation.

A gradual absorption of the dura mater from within, and consequent thinning and perforation, is occasionally observed to follow the growth of tumors, from the brain, or inner membranes; it may also result from an unusual increase in the size and number of the Pacchionian bodies. Of these we shall have occasion to speak, when considering the relations of the arachnoid.

Little is to be said concerning heterologous products occurring in the dura mater; the same cause which accounts for the comparative rarity

Fig. 113



A portion of dura mater, exhibiting a mass of bone-like substance of low conical form, attached to the side of the falx cerebri. The patient, 28 years old, had been subject to severe headaches from boyhood. A fortnight before death acute headache supervened, followed by delirium, partial paralysis, and insensibility. There was copious effusion of lymph in the cerebral membranes and ventricles.

* Lateral view of the same. St. Bartholomew's Museum. Series vi. No. 46. Catalogue, vol. i. p. 201.

of idiopathic inflammation in this membrane, suffices to explain why it is less frequently the seat of adventitious growths, than the other envelops of the brain. Fibroid tumors are not unfrequent in connection

with the dura mater, from which they are more prone to arise than from other structures within the cranium; according to the prevailing law, that the physiological properties of a structure are liable to influence the morbid growths springing from it. In many instances these tumors would come under the head of hypertrophy, rather than of heterologous products.

Both on the internal and external surface of the dura mater we frequently meet with small laminæ of bone, which, in many instances, may be owing to chronic inflammatory action, of which no other trace is left. Some writers deny that the ossifications found on the inner surface of the dura mater are the products of this membrane; they view them rather as growths belonging to the arachnoid, an opinion to which we demur, both on account of the position in which these bone deposits are found, and from the general endowment of periosteal tissues, to which class the dura mater undoubtedly belongs, to generate bone. These ossifications, which are easily separated from the dura mater, and are generally met with along the falx, must not be confounded with the hypertrophy of the frontal and parietal bones, said to occur during pregnancy, and hence termed, by their discoverer, Rokitansky, puerperal osteophyte. The most remarkable instances of genuine ossification, or of osseous deposit in the dura mater, are occasionally met with in chronic hydrocephalus, where it seems to indicate an effort of nature to afford extra protection to the diseased brain. A delineation of this affection is given in Dr. Bright's medical reports.

Cysts of a lipomatous character are occasionally found attached to the dura mater.

We have already seen that the dura mater is frequently subject to secondary inflammation, owing to the extension of scrofulous or tubercular inflammation from adjoining bones; it may thus, as also by extension of tubercular disease from the cerebral tissue, become the seat of tubercular deposit; but the primary deposit of tubercle in the dura mater is, probably, never seen. Tumors of a cancerous nature more frequently grow from the dura mater; and, although the term "fungus of the dura mater" may occasionally have been falsely applied to carcinomatous formations within the cranial bones, still, morbid anatomy supplies numerous instances of undoubted cancer of the dura mater. It occurs either in the form of an infiltration of the membrane; in consequence of which the dura mater becomes thickened, and may, by degrees, communicate the carcinomatous infection to the arachnoid, or the bones; or it assumes the form of a rounded tumor, which generally consists of medullary cancer, and pushes its way through the adjoining bone. It is commonly very vascular, and generally occupies a situation near the vertex. When it forces its way through the osseous parietes, these form a ring round it, and the external table of the cranial bone will present a smaller opening than the internal table. Its growth becomes very much more rapid when it has passed the bounds in which it is first kept by the skull, and the soft parts soon become involved and perforated.

The dura mater rarely exhibits any malformation or defect, beyond those spoken of, except that the falx is occasionally found cribrated,

and that in old people the dura mater presents slits in the vicinity of the longitudinal sinus, varying from two to four lines in length, through which the Pacchionian glands have forced their way.

The records of the Pathological Society of London contain the account of a very rare defect of the falx cerebri, exhibited by Mr. Shaw.[1] Dr. Bright[2] also gives a similar case, in which no trace of the process was visible anterior to the tentorium, and it was assumed that the defect, which occurred in a lady thirty years of age, had existed from birth.

[1] Reports of the Pathological Society, 1847–48, p. 178.
[2] Medical Reports, vol. i. p. 150.

CHAPTER VIII.

THE ARACHNOID AND PIA MATER.

THE traditional doctrine of anatomy, that the arachnoid is a serous membrane of the same character as the pleura or pericardium, has found powerful opponents in Doctors Henle and Kölliker, who have shown that the external lamina is nothing more than an epithelial layer investing the dura mater. It must also be remembered that the arachnoid is entirely dependent for its supply of blood upon the pia mater; and that, consequently, in health as well as disease, the condition of these two structures necessarily bears a very intimate mutual relation. The pathologist has, instinctively as it were, adopted this view; inasmuch as the term meningitis is generally understood to comprise inflammation of the arachnoid and the pia mater, to the exclusion of inflammation of the dura mater, which is not implied in the name; while arachnitis is commonly used to designate disease of the arachnoid, as well as the pia mater, although etymology would not sanction such an interpretation. The best authorities of our own country, on subjects connected with the pathology of the brain and its membranes, as Doctors Watson, Abercrombie, and Bright, are opposed to over-refinement in these distinctions, and are inclined to deny the limitation of disease to one or the other of the structures under consideration. It certainly is more practical, and less likely to mislead the student, if we treat of the morbid appearances of the arachnoid and pia mater under one head, pointing out as we go on those cases and diseases in which the one or the other may appear exclusively or mainly implicated. The arachnoid depends solely for its supply of blood upon the pia mater, which may, therefore, fairly be regarded as its matrix; in fact, it is very doubtful whether the former contains any bloodvessels of its own; and, although it is commonly assumed that the arachnoid possesses the power of secretion, this would seem rather to be of a mechanical than of a vital nature.

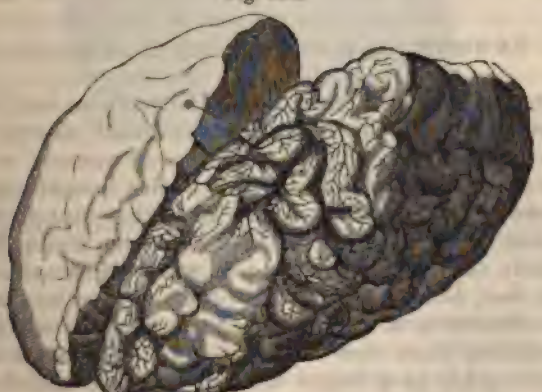
Though the traces of active congestion or hyperæmia of the arachnoid are not visible to the eye after death, we constantly meet with changes in the physical characters of the membrane which are the result of increased action, though the history of the individuals fails to show the occurrence of actual inflammation; opacity and thickening of the arachnoid, especially on the surface of the hemispheres, is so frequent after the middle period of life as almost to merit being classed under the changes of involution, but it is most marked in habitual drunkards; in delirium tremens, it is often the only disorganization to be found in the cranial cavity. This opacity is commonly accompanied by more or less serous

effusion, which fills the sulci, and raises the membrane from the surface of the brain. Instead of being transparent, and allowing the vessels of the pia mater to shine through, the surface looks milky to a greater or less extent, and more particularly on the surface of the hemispheres. Owing to the obliteration of the sulci by the serum, the affected portion of the cerebrum often looks as if the convolutions were compressed, but on removal of the serum the cause of this appearance at once becomes evident. The subarachnoid effusion may be independent of any affection of the arachnoid, and be solely due to congestion of the pia mater; we should then find no adhesion between the tissues, the arachnoid retains its transparency, and the fluid is more prone to follow the laws of gravitation, and form pouches at the dependent portions of the organ.

It may fairly be questioned whether the subarachnoid fluid is, in any way, due to the action of the arachnoid membrane. Whenever we find serum between the layers of the arachnoid, and in the ventricles, to adhere to the received terminology, it is right to refer it to that membrane; but the vicinity of a congeries of vessels, and the known laws of transudation, certainly favor the view that subarachnoid effusion is attributable to the pia mater only. Congestion of the vascular network contained in this membrane is extremely frequent, though we are more frequently left to infer it, during life, than that we find it after death. It is the very nature of congestion to disappear *in articulo mortis.* Still, there is a sufficient number of affections in which the vessels of the pia mater have been shown to be gorged with blood, without any further accompanying morbid conditions of the adjoining structures; thus, we meet with it in cases of pertussis, of fever, of capillary bronchitis, or disease of the heart. The congestion may, in the latter case more particularly, attain an extreme degree, so that, the blood not having room in the veins, we find dark venous-colored blood even in the larger arteries.

There is no doubt that, occasionally, the subarachnoid fluid is attri-

Fig. 114.



Subarachnoid effusion on the upper surface of the anterior lobe, causing an apparent obliteration of the interstices between the convolutions, and accompanied by increased vascularity.

* Enlarged Pacchionian bodies.

butable to cadaveric changes; it is, therefore, necessary to be circumspect in at once attributing its presence to antecedent morbid action. The amount and position, and, more particularly, the concomitant appearances of the pia mater and arachoid, must assist us in determining the question in the individual case.

It is not yet decided whether the small nodules, which are found in almost every brain, but become more numerous with advancing age, and have received the name glandulæ Pacchioni,[1] are pathological products, or normal constituents. A superficial examination suffices to show that they are not what their first discoverer assumed them to be, conglobate lymphatic glands. They consist of an irregular fibrous network, containing some albuminous granular matter, and generally occupy the vicinity of the mesial line of the surface. They often cause perforations of the dura mater, and may thus appear to belong to this membrane, when the brain is taken out in all its envelops. Their development, at times, is so considerable as even to induce absorption and thinning of the skull-cap. The term arachnoid granulations, applied to them by Louis, is probably as correct a designation as any that has been given them, and we should be inclined to attribute to them no higher importance than that belonging to warty indurations on the surface of the body. Luschka, who has especially investigated the Pacchionian bodies, confirms this view, and describes them as cactus-like projections from the arachnoid, of a fibrous organization, vascular, and covered by a scanty epithelium. He regards them as normal constituents of the membrane, but states that the hypertrophy to which they are liable may be the cause of death by the pressure they occasion.

The arachnoid is occasionally found to present an unctuous sensation to the finger, without any marked morbid changes being discoverable in its vicinity; though there is generally considerable disease in some part of the brain, which would account for a change of nutrition and secretion in a membrane like the arachnoid. This is the case in four out of five cases in which Dr. Bright applies the term to the arachnoid.

HEMORRHAGE INTO THE ARACHNOID.

We come now to the consideration of hemorrhage into the cavity of the arachnoid, a subject which presents a peculiar interest, from being the form of cerebral sanguineous effusion most common in childhood; the reverse condition obtains in adult life, when hemorrhage within the cerebral tissue is the prevailing form. In neither case do we generally succeed in tracing the mouths of the vessels from which the discharge has proceeded; though it is easier to do so than in most other hemorrhages. In fact, Dr. Watson[2] lays it down as a rule, that, while the hemorrhages occurring in the lungs, and other organs of the body, are due to exhalation, those occurring within the cranium are attributable to the actual rupture of a larger vessel. We have already alluded to

[1] Ant. Pacchioni Dissert. Epistolaris de Glandulis Conglobatis Duræ Meningis. Romæ, 1705.

[2] Lectures on the Practice of Physic, &c. vol. i. p. 494.

one variety of hemorrhage occurring in the tissues of the head in the new-born infant, which we stated to be the result of the mechanical pressure exerted during parturition; the same cause may give rise to arachnoidal effusion, which then commonly and speedily proves fatal. In this case, we find a larger or smaller quantity of fluid blood investing the upper surface of the brain; but, if the child survives the immediate shock of the apoplectic seizure, the effused blood will undergo that series of changes, which indicate the tendency of the natural processes to restore the parts to their normal condition. The first step is the formation of a coagulum; the fluid portions are first absorbed, then, by degrees, the coloring matter passes through various changes till it also disappears, and we then find, in the place of the original coagulum, a membranous formation, which is more or less organized, and may be shown to contain a capillary system of its own. It is a singular act in the pathology of the disease, to which MM. Rilliet and Barthez were the first to draw attention, that, unlike cerebral hemorrhage in the adult, it rarely gives rise to paralysis in the child; a circumstance explained by Dr. West,[1] upon the principle of the blood in the latter instance being almost invariably effused into the cavity of the arachnoid, in consequence of which, the effects of the pressure act more uniformly upon all the contents of the cranium. The changes in the effused blood may, however, assume another form than the one above described, and it is important to be aware of the circumstance, as it may stimulate hydrocephalus. Instead of the serum being absorbed,

Fig. 115.

This drawing represents a portion of dura mater divided from above, and showing a sac which was filled with coagulated blood. The coats of the sac presented nearly the same thickness as the dura mater.—St. Bartholomew's Museum. Series vi. No. 25.

it may become inclosed in a false membrane, and remain as a persistent sac, exerting an amount of constant pressure upon the subjacent brain, sufficient to cause the flattening of the surface, and to induce, as they generally do, a considerable impairment of the intellect. When these

[1] Lectures on the Diseases of Infancy and Childhood. By C. West, M. D., London, 1848, p. 40.

cysts are once formed they have a great power of passive resistance, and rarely diminish in size.

Three interesting and instructive cases of superficial hemorrhage occurring in the adult, are given by Dr. Abercrombie;[1] and we also find a few instances recorded by Dr. Bright.[2] In the former, nothing was found either in the brain or the other viscera to account for the effusion; in one of the cases contained in the *Medical Reports* there was a hypertrophy of the heart; in another, the hemorrhage was due to the rupture of a small aneurism; in a third, no lesions were found; and in two others the hemorrhage was attributable to a fall, and the viscera do not appear to have presented any disorganization.

ARACHNITIS.

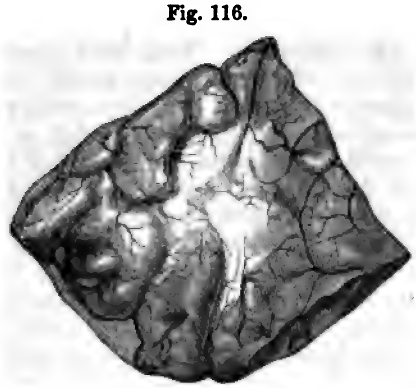
Arachnitis is a term used synonymously with meningitis, to designate inflammation of the arachnoid and the pia mater. The nosologist may at the desk draw numerous fine distinctions, and classify symptoms so as to produce a uniform system of morbid processes; but nature does not bind herself to laws of this description. This remark applies forcibly to the attempts made to dissever inflammation of the visceral plate of the arachnoid from inflammation of the subjacent pia mater. Whether in the course of pathological research we shall be justified in establishing more systematic divisions than we are now able to do, is not the question; but we deem it especially our duty, throughout this work, to place before the student of pathology facts which he may recognize in the dead-house, from their having been previously observed by trustworthy inquirers, rather than to show him a maze of systems which have long been the opprobrium of scientific medicine. The subject immediately before us is a stumbling-block to the practitioner, as much or more than it has been to the pathological anatomist. The former has allowed early and traditional impressions of the necessity of employing powerful antiphlogistic treatment in all cases where heat of surface, restlessness, a tendency to, or actual convulsions, the drawn-in thumb, and other well-known symptoms, seemed to indicate inflammatory action; and many a child has been sacrificed at the altar of school-science, where the avoidance of injurious influences, or a strengthening regimen, would have succeeded in restoring the patient. Similar observations apply to much of our treatment in cerebral affections of adults, in whom cephalic symptoms are, even at the present day, too uniformly looked upon as the legitimate excuse for the application of our entire antiphlogistic apparatus. These remarks have suggested themselves by the great difference in the causes and course of several diseases, which are each due to, or connected with, inflammatory conditions of the pia mater and arachnoid; and there is also one disease which must be classed with the former, which with symptoms resembling those of meningitis leaves no

[1] Pathol. and Pract. Researches, &c. p. 242.

[2] Medical Reports, &c., p. 266. See, also, Mr. Prescott Hewett's paper in the Medico-Chir. Trans., vol. xxviii.

trace of inflammatory or morbid action. Under the first head we class simple meningitis and tubercular meningitis; under the second, we allude to hydrencephaloid disease, to which Marshall Hall[1] first drew attention, and the analogue of which is presented to us in the adult, in that form of cephalic disease, termed somewhat indefinitely, serous apoplexy. In a case of simple acute meningitis, three points especially deserve attention: the vascularity of the membranes, the adventitious membrane formed between the arachnoid and the pia mater, and the effusion of serum or pus in the same position. We may, according to the stage and duration of the disease, meet with either of these appearances, or they may be combined in the same individual. If the patient has died in the early stage of the disease, we find, on removing the dura mater, that the subjacent membrane shows a great increase of vascularity, which may be so intense as almost to resemble the effusion of blood; the eye and the touch will, however, speedily detect the real nature of the discoloration. The congestion spreads more or less over the surface, or appears in circumscribed patches; on removing a portion of the arachnoid, we shall find the congested vessels dipping down with the pia mater between the convolutions. If effusion of lymph has taken place, a membranous expansion will be found here and there to intervene between the two meninges, causing a sort of marbled appearance, or bands stretching from one convolution to another. The effused lymph attains the thickness of a wafer and more, and most commonly occupies the upper portion of the hemispheres. The lymph itself dips down into the convolutions, and presents the same variations of density and consistency that this product of inflammation offers elsewhere in proportion to the date of its effusion. Some serous effusion beneath the arachnoid of the

Fig. 116.



Portion of upper cerebral hemisphere of a young woman, aged 27, with purulent effusion under the arachnoid: there were two yellow symmetrical patches, one on each parietal surface, concealing the subjacent convolutions.

base, especially about the optic chiasma, which causes the part to resemble the appearance of jelly, and a small amount of similar fluid (from two drachms to an ounce) in the ventricles, are often found in this form

[1] On Diseases and Derangements of the Nervous System. London, 1841.

of arachnitis, though it is by no means a necessary accompaniment. The brain, in these cases of simple meningitis offers no appreciable derangement of structure, though the symptoms during life may have shown very manifestly that its functions were involved. The formation of pus in the course of arachnitis is not an occurrence of frequency, but it is necessary to bear in mind that it is a pathological fact. A remarkable instance of this occurred under our observation recently, at St. Mary's Hospital, in a young woman in whom sudden and unexpected coma supervened, and terminated, after thirty-six hours, in death. She had previously suffered from otorrhœa; but on her admission gave no evidence of cephalic disease, nor was any direct connection traced after death between the affection of the ear and the meningitis which was found to have caused her death. Here two yellow patches were discovered on each parietal surface of the brain, owing to an accumulation of pus spread out under the meninges; the microscopic examination of the fluid satisfactorily demonstrated the characters of pus.

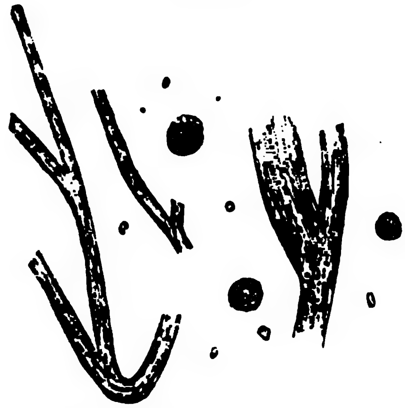
Cases of meningitis are now and then met with, in which the arachnoid appears perfectly transparent and normal, while there is vascularity of the pia mater with subarachnoid effusion. We should here be inclined to assume an idiopathic affection of the pia mater, and it cannot be denied that the evidence in favor of primary and independent disease in that membrane, is stronger than any arguments adducible in favor of the same disposition in the arachnoid. We cannot blind ourselves to the fact that the latter differs much in its behavior from the serous membranes of the thorax and abdomen, in the rarity of inflammatory effusion occurring within what we must term the sac of the arachnoid, if we continue to look upon it in the same light as a serous membrane. It is highly desirable that anatomists should settle its normal relations, in order that the deviations occurring in morbid processes may receive the correct and proper estimation. Thus, with regard to the effusion occurring within the ventricles of the brain, which are commonly taught to possess an arachnoideal lining, it evidently offers many relations different from the arachnoideal effusions occurring on the surface of the brain; nor is it quite intelligible why the secretion into the cavity of the ventricles should so rarely be found to communicate with the superficial arachnoideal space. Kölliker,[1] whose profound knowledge of anatomy and physiology renders him an authority in the matter, says: "Those who state that the arachnoid lines the ventricles of the brain, and the processes of the pia mater contained in them, suppose a thing that is impossible, viz., that the arachnoid passes through the pia mater, and invests the surface of the plexuses, which is actually an internal one."

If we examine the inflamed pia mater under the microscope, we shall find the smaller vessels studded with exudation matter, in the shape of minute oily-looking vesicles; we have seen it as in the adjoining drawing, so bounded by the outline of the vessel, that it seemed to lie within its coats; there is no *à priori* reason why it should not be formed in the vessels. We also see numerous so-called inflammation corpuscles,

[1] Mikroskopische Anatomie von Dr. A. Kölliker, 1850, vol. ii. p. 501.

which would appear rather to be a peculiar aggregation of the exudation matter than distinct formations; they are sometimes possessed of a delicate envelop, at others they are devoid of it; they may be appro-

Fig. 117.



Meningeal vessels invested and surrounded by exudation matter, and glomeruli or exudation corpuscles, from a young man, aged 34, who died in consequence of injury to the head, followed by meningitis; magn. 270 di.

priately compared to a mulberry—they generally, also, offer a brownish tint. We owe the knowledge of these corpuscles to Professors Gluge

Fig. 118.

Fig. 119.

Further specimens of the microscopic appearances of the vessels in meningitis.

and Bennett; the former was the first to show their connection with inflammation generally, the latter drew attention to their presence in inflammatory affections of the brain and its membranes.

Much confusion has arisen from the misapplication of the term hydrocephalus, as it has been used to designate a variety of diseases, simply on account of their resembling one another in a comparatively

accidental feature; and we would, therefore, follow in the steps of those authors who limit the term to the dropsical effusion of serous fluid within the cranium, unaccompanied by marked symptoms of inflammatory action during life. We have already seen that an accumulation of serum beneath the arachnoid, or within the ventricles, is a common feature in both acute and chronic meningitis; and though an affection of serious import, we have abundant evidence of the value of therapeutic proceeding in arresting and completely removing the disease and all its effects. Not so with what is commonly called chronic hydrocephalus, or what ought exclusively to receive the name of hydrocephalus, while certain forms of meningitis should designate the disease acute hydrocephalus; nothing is more liable to mislead the student, or to perpetuate error, than a want of precision in our nomenclature. We must never forget that the effusion of serum is only a product of morbid action; and that inflammation, mechanical obstruction, anæmia, blood-poisoning, scrofulous cachexia, diseased conditions essentially differing from one another, may each of them give rise to a secretion of fluid into serous cavities. It is erroneous, and likely to lead to the most injurious practice, if we apply a name to a symptom, by which it becomes identified with the most opposite diseases.

Whether we look upon the arachnoid as a serous membrane or not, and whether or not we continue to consider the lining of the ventricles as a prolongation of the external arachnoid, there is no doubt of the close resemblance between the structure of the latter, with this difference, that while the superficial arachnoid overlays the pia mater, the lining of the ventricles is in direct opposition with the gray matter of the brain. In examining the pathology of the brain, we shall have occasion again to allude to the ventricles, but it seems advisable to advert to some changes which occur in them at this part of our inquiry. Here, as in the superficial arachnoid, we see no traces of active congestion, even where we have the undoubted evidence of inflammatory action. Hemorrhage into the ventricles is invariably the result of injury to or disease of the brain, and the effusion of blood into them is stated by Dr. Bright to be the most rapidly fatal of any kind of apoplexy. It has already been mentioned that in meningitis it is common to find an increase in the ordinary amount of secretion of the ventricular fluid; but while this may be measured by drachms, the fluid accumulating in hydrocephalus reaches to several pints, and is commonly the result of defective action, not so much in the membrane as in the cerebral and general circulation. Though we fail to find a congested state of the vessels in the ventricular lining membrane, it often presents an indurated, thickened, and granular condition, indicative of previous inflammation. The septum lucidum, and the commissura mollis, are generally involved in the morbid processes occurring in the ventricles: the former is more particularly liable to suffer in chronic hydrocephalus from distension; and yielding to the mechanical pressure, it becomes perforated, and allows of free communication between the two cavities. The granulations of the arachnoid occasionally become prominent, and even pediculated; at other times the inflammatory product is rather of an adhesive character, and the opposite sides of the

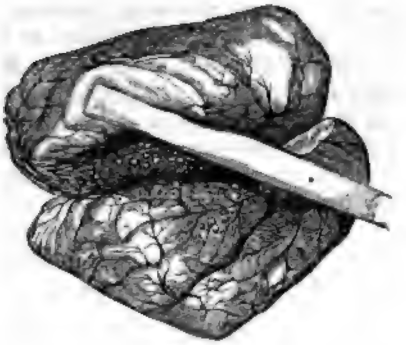
ventricles, especially the corpora striata, may then become agglutinated to one another.

It must be considered as one of the great goals which this and future generations of scientific inquirers have to pursue, in how far these and similar changes in the membranous or parenchymatous structures are to be attributed to dyscrasic conditions. The combined modes of research represented by the microscope and the test tube, must assist in solving many of the problems that yet puzzle the physician; and if there are various cachectic conditions differing essentially in their character, of which we have no doubt, we must hope to succeed eventually in demonstrating them more palpably than has yet been done, both in the living and the dead subject. That the rheumatic and gouty diathesis should produce their definite lesions within the cranium, as well as externally, can scarcely be denied; but we do not possess sufficiently satisfactory evidence. Purpuric spots are occasionally seen on the arachnoid, indicating a scorbutic crasis; but none of the crases have hitherto been shown to have such decided traces within the cranium as the scrofulous or tubercular. The frequency of meningitis in childhood is in a great measure due to this fact; and to it we may also in a measure attribute the great prevailing fatality, owing to the misconceptions that the term inflammation gave rise to; and which, therefore, seemed to necessitate antiphlogistic treatment of as active a character as if we had to deal with sthenic inflammation.

TUBERCULAR MENINGITIS.

The deposit of tubercle on the pia mater of the brain occurs in the shape of small miliary granules, resembling the Pacchionian bodies in

Fig. 120.



Deposit of tubercular matter in the Sylvian fissure of the brain of a child, aged 19 months, who died ten days from the first appearance of head symptoms, which were treated antiphlogistically. The white circular spots represent the tubercles, which were surrounded by highly congested bloodvessels. The white deposit examined by the microscope showed granular matter and granular corpuscles varying in size.

appearance, but differing from them both in their site and in their microscopic relations. They are not seen on the free surface of the arach-

noid; and, in fact, seem in no way connected with this membrane; a point which establishes a marked difference between it and the serous membranes of the thorax and the abdomen. They are found most frequently deeply within the Sylvian fissure, in the convolutions of the brain, and at the base; they are of the size of pins' heads, and appear in the form of gray granulations, imbedded among a vascular network; they are very rarely found upon the cerebellum. A careful examination is necessary, to prevent their being overlooked; but in a therapeutic point of view, it is most important that their presence should be duly appreciated, and that they should be taken as an indication of a state of the blood, requiring a different line of treatment from what ought to be adopted in meningitis, unconnected with the pathological condition of the fluids that tubercle implies. It is only by the aid of the microscope that we have been able positively to determine the real nature of these granulations, for they closely resemble a mere puckering of the membrane; seen under a power of three or four hundred diameters, their elements resolve themselves into the corpuscles peculiar to tubercle, possessing a faint outline, with granular contents, and surrounded by granular matter. The tubercular corpuscles must not be confounded with epithelium, which may be seen under the same field, and which is to be distinguished by the nuclei it contains, within which, again, one or two nucleoli are visible.

This form of meningitis is generally met with in connection with or secondary to tuberculosis of other organs; though we see it occasionally in the idiopathic form, as in the instance from which our delineation was taken, where no tubercular deposit was found in any of the viscera beside the brain. In the former case, the tubercular deposit may take place so insidiously as to offer no marked inflammatory symptoms during life; and it is only on the dissecting-table that the physician becomes aware of the cerebral disease. It must, therefore, be borne in mind that scrofulous children have a tendency to become afflicted in this manner; and it is an additional reason for watching them with care, and avoiding such debilitating measures as would be likely to encourage the dyscrasia. It is not our province to enter into the consideration of nosological or therapeutic inquiries, but we cannot avoid remarking that the frequency of tubercular deposit in the pia mater in children is a strong argument against those powerful depleting measures which inflammatory symptoms presenting themselves in the head of a young subject, are generally calculated to provoke.

HYDROCEPHALUS.

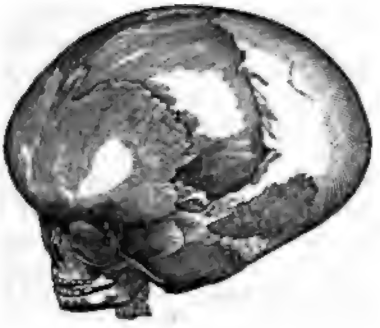
The irritation of the tubercular deposit is very liable to induce the secretion of serum, either beneath the arachnoid membrane, or in the cavities of the brain, and this leads us to the consideration of that formidable disease of childhood, hydrocephalus.

The etymology of the term indicates the main feature that characterizes the morbid condition, and, provided its application be limited to the class under consideration, it is not likely to mislead the practitioner, as

it implies no theory. It is truly a dropsy of the brain, and it is an affection which, like dropsies in other parts of the animal economy, is favored by a relaxed and soft condition of the surrounding tissues; the anatomical peculiarities of the fœtal and infant head at once indicate a probable reason why it should commence at those periods; while the mal-nutrition upon which it is based, and the further impairment of the important organ in which it occurs, is a sufficient ground for explaining its great fatality, and the infrequency of the child affected with it surviving to reach manhood. Instances are, however, on record, of individuals in whom the parts accommodated themselves to the morbid effusion, and life was prolonged far beyond the period of childhood. The most celebrated case of this kind is that of James Cardinal,[1] who attained the age of twenty-nine, though he had been hydrocephalic from within a fortnight after birth; the circumference of his skull measured at the period of his death thirty-two inches and a quarter.

The accumulation of serum frequently commences in the ventricles, which are the most ordinary seat of hydrocephalus, or on the surface of the brain, during the last months of fœtal life; it may become a circumstance for the consideration of the accoucheur during parturition,

Fig. 121.



A hydrocephalic skull from a girl aged 11 years: the enlargement of the skull is effected by its elongation, and by the depression and hollowing of its base. An increase of width appears to have been prevented by the premature and complete closure of the sagittal suture. The coronal suture, and that between the frontal bone and the suture, also of the splenoid, are wide open. The superior walls of the orbits are pressed downwards. The bones generally are thin and light. St. Bartholomew's Museum, sub series x, 2.

not so much from the increased size of the head presenting an obstacle, because there is even a greater compressibility than usual, but from the head being so soft as not to offer a sufficient fulcrum for the labor pains to act upon, or, in case of operative interference, for the instruments to obtain a sufficient purchase. The child may, however, be born apparently without a blemish, though it does not thrive well, and a few weeks after birth, or at least within the first three years of life, the head appears to increase unduly in size, and the ossification of the fontanelles is retarded. Dr. West states,[2] that out of fifty cases, symptoms of hydrocephalus were observed in forty-six before they were six

[1] Dr. Bright's Medical Reports, p. 481.
[2] On the Diseases of Infancy, 1848, p. 84.

months old, and in twelve of these the malady was congenital, and that in nineteen more it came on before the completion of the third month. The accumulation of fluid may amount to as much as ten pounds. The increase in the quantity is greatest during the first months, and diminishes or even remains stationary if the child survives; the circumference of the head corresponds to these relations; thus, it is not uncommon to find the circumference to attain as much as twenty-six inches and more, during the first three months, and to vary but little subsequently.

The immediate pathological effect of this secretion is to compress the parts within the skull, and to distend and prevent the due ossification of the cranium; the frontal bone is made to protrude, and the parietal bones, from their yielding character, bulge out considerably on each side, while the intervening soft parts present fluctuation. Ossification takes place, though irregularly, and at a much later period, and we have already seen that a succedaneous deposit of bone is occasionally effected in the dura mater. The sutures of the cranial bones are not formed as in the healthy skull, but are less serrated, and therefore more ready to yield to pressure from within, which they occasionally do, after they have closed, in the event of a sudden increase of the fluid. The ossification does not proceed with the normal regularity, but numerous centres form, and thus we meet with more or less ossa triquetra in the line of the sutures. In an almost similar ratio, we shall find the brain altered in its relations. If the accumulation has been confined to the ventricular cavities, the entire brain will be distended, and its tissue is generally found softened and pultaceous, from the fluid having infiltrated the cerebral tissues. The distension may be so excessive as to reduce the hemispheres to the thickness of a sheet of paper; but more commonly the parietes give way, and allow the fluid of the two sides to combine; and the pressure may then, as in the case of Cardinal, separate the hemispheres, and unfold them, "like the leaves of a book."

Occasionally the fluid is limited to one side, and thus gives rise to an unilateral distension or obliquity. When the superficial arachnoid is the seat of the dropsy, the brain may, at first sight, appear altogether deficient; but, on raising the sac, we shall find it pressed downwards and forwards, and presenting a state of extreme atrophy.

It is usual to meet with some abnormal conditions of the vessels and the membranes in hydrocephalus, but we are unable to determine the relation that they bear to the morbid product—whether they are to be considered in the light of cause or effect; though we find both Dr. West and MM. Rilliet and Barthez agreeing that hemorrhage into the cavity of the arachnoid may occasionally be the *fons et origo mali.* Rokitansky attaches more importance to hemorrhage, as occurring in the course of the disease. A thickening and opacity of the lining membrane of the cyst, with granular deposits, are the most common post-mortem appearances found in the arachnoid, while occasionally the formation of a new membrane, and the deposit of fine granular matter, causing a roughness of the surface, present themselves to the anatomist. The occurrence of the latter suggests that there has been intercurrent, subacute inflammation, and should warn us to remove all causes

of irritation from an individual thus circumstanced. The vessels subjacent to the arachnoid are frequently enlarged, varicose, and congested.

The fluid of hydrocephalus is of a limpid and transparent character, or more or less yellowish and opaque; of a specific gravity rather lower than that of the serum of the blood, and containing, therefore, a smaller amount of solid constituents. When there is any opacity, this is found to be due to granular and nucleolar matter, mingled with epithelial debris. The fluid, when tested by heat and nitric acid, presents the characters of a solution of albumen, and is found to contain chloride of sodium, soda, and traces of salts of lime and potash; and also, according to Dr. Bostock's investigation, urea. The results of his analysis of the hydrocephalic fluid of Cardinal are embodied in the following table:—

Sp. gravity 1011.38

Water	982.6
Albumen	6
Chloride of sodium	7
Soda	1.4
Urea and osmazome	8
Sulphuric acid, lime, potash	a trace
	1000.0

The fact of urea being found in hydrocephalic fluid has not met with any attention on the part of other pathologists;[1] but if it should prove to be the rule, it places this disease on a par with the uræmic cases, which are intimately connected with certain forms of renal disease, to which Drs. Bright and Christison first drew attention. The state of the kidneys is a point which merits more consideration in this disease than has hitherto been devoted to it, or we may say in infancy generally. It is to the generic development of an affection like hydrocephalus that we must look for the attainment of a means of cure; for its products, though interesting to the pathologist, are unfortunately not of a character to yield either to the vis naturæ or to medical treatment. From the early appearance of its symptoms it especially challenges our attention to the state of health of the progenitors; and though in many of the recorded cases it is expressly stated that the mother's constitution had been sound, we must bear in mind how vague such data generally are, and that the hereditary influences are as intimately connected with the father as the mother. We know more of the relation existing between hydrocephalus and tubercle; for the two are very commonly found associated. Scrofulous children very frequently present a very considerable amount of serous accumulation within the ventricles, without even having manifested any symptom of cerebral disease.

We have spoken of hydrocephalus throughout as of a disease of childhood, and it is to early life that it mainly belongs. Several cases are, however, on record of the affection having supervened in the adult when there was no trace of previous cerebral disease. If it be found

[1] Dr. Garrod has stated to us in conversation, that he has also found urea in hydrocephalic fluid.

that the diathesis upon which the disease is engrafted belongs equally to infancy and manhood, we shall easily be able to explain why the more prominent symptom of dropsical accumulation does not present itself frequently in the adult. The firm connection of the cranial bones, and the compression which they consequently exert, offer a mechanical impediment to the effusion, which can only take place at the expense of some other contents of the skull. The very congestion of the veins, which for instance exists in cases of uræmia, would militate against it.

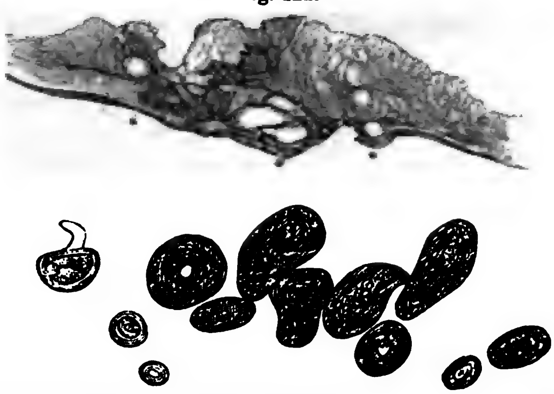
Several instances of an accumulation of serum in the cavity of the arachnoid or in the ventricles, and not offering any marked traces of active hyperæmia or inflammation occurring in advanced life, may be found in the works of Professor Gölis, Dr. Baillie, and Dr. Watson. They have received the name senile hydrocephalus, and such are the cases to which the nosological term of serous apoplexy ought to be restricted. The fact of Dean Swift having died of this affection, may serve to fix the subject in the memory. The immediate cause to which this may be most justly attributed is an atrophic condition of the brain, which gives rise to a vacuum, and hence to a discharge from the vessels, of that part of the blood which most readily transudes the coats; the effused serum is peculiarly clear, and its quantity varies, according to the extent of cerebral atrophy, from two to six ounces. When we examine into the morbid anatomy of the brain, we shall discuss the origin and causes of atrophy; we have here only to do with one of its products; but it is apparent that in the present instance, as well as in the hydrocephalus of infants, the effusion is a symptom of a deeper-seated malady, and not truly an idiopathic affection. In senile as well as infantile hydrocephalus, the soft commissures, the septum, fornix, and adjoining parts, are commonly found softened; but it is not always easy to form a positive opinion, whether this is a primary or secondary condition. Before dismissing this subject, we have to advert to a form of congenital hydrocephalus which has received the name of hernia cerebri, in which, owing to a deficiency in the cranial walls, a portion of the brain and its membranes are protruded. It is analogous to the spina bifida.

CHOROID PLEXUS.

The venous rete mirabile of man, known by the name of the choroid plexus, appears in a measure to possess a vitality independent of the membrane, the pia mater, of which it is an appendix. It is impossible to doubt that it plays a most important part in equalizing and balancing the circulation within the cranium, within the limits of health, and that equally its peculiar relation to the cavity in which it is suspended must give rise to important variations in disease; the physical laws of exosmosis and endosmosis may, without any stretch of hypothesis, be supposed to operate with peculiar vigor, and it is not unreasonable to assume in the choroid plexus a powerful agent of secretion and absorption. The amount of blood in the plexus found after death varies much —at one time it is full, and the vessels stand out in relief; at others it

is collapsed, and contains scarcely enough blood to color it. There is no necessary relation between the amount of vascularity or exudation, and the congestion or inflammation of the pia mater; thus, we may find the surface of the brain covered with a highly vascular pia mater, while the choroid plexuses present an exsanguine appearance; a circumstance which rather confirms our view of the physiological character of the latter. The greater density of the coats of the vessels in the choroid than in other parts, may account for the rarity of their being the source of hemorrhage; indurated yellow bodies are, however, occasionally found in them which are referable to former effusion. The morbid appearances most commonly found in the plexuses are round or oval bodies of a yellowish tinge, apparently formed of concentric laminæ, which only become more apparent on the addition of acetic acid. They are ordinarily microscopic, but are often found in considerable numbers, and occasionally accumulate into masses of the size of a pea or small nut.

Fig. 122.

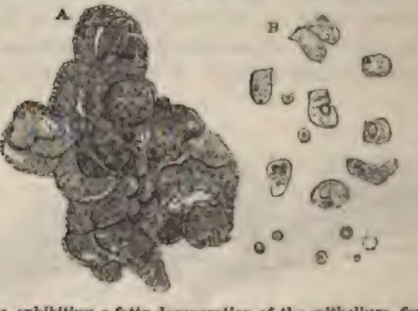


The upper figure represents a choroid plexus with several small tumors at * * *, supposed at first to have been tubercular; they proved to consist of aggregations of concentric corpuscles, cholesterin, and pure oil, united by areolar tissue; the concentric corpuscles which are shown below the plexus are magnified 100 diam.

It is evident that they are phosphatic connections enveloped by layers of organic material. Acetic acid acts very slowly upon them, destroying their opacity, but not altering their configuration. Muriatic acid destroys their opacity by removing the phosphate of lime, and leaves transparent rings of animal matter. They are not found in early infancy, but occur so frequently in advanced life that they almost appear to be a normal constituent from their behavior with reagents. Virchow calls them corpora amylacea, but the term concentric corpuscles, suggested by Dr. H. Jones, is more appropriate. Similar formations are met with in other parts of the body: thus, we have seen them in an adventitious membrane formed in the kidney. Cysts of the choroid plexus are more frequently mentioned by authors on account of their being visible to the naked eye; they are minute, transparent vesicles, varying in size from a poppy seed to a small pea; and we generally meet with them in brains, in which we also find evidence of inflammation

of the lining membrane of the ventricles. They sometimes contain a milky fluid, and may present incrustations of a sabulous character. They have been erroneously regarded as hydatids, but there is no evidence of their belonging to this class of parasitic formations; they would rather seem to be due to a condensation of the epithelial covering of the plexus, and an accumulation of fluid beneath it, limited by an effusion of plastic matter. An hypertrophy of the epithelium, which covers the choroideal vessels, is very commonly observed in advanced age—though we have not been able, as yet, to trace any definite relation between this condition and certain forms of disease. The choroid

Fig. 123.



Portion of a choroid plexus, exhibiting a fatty degeneration of the epithelium, from a female aged 45, who for 14 days previous to her death was subject to convulsive fits and various cerebral symptoms. No morbid condition was observed in the brain, but considerable deposit of oil in the choroid plexuses; in one, there was a small lump of fat. A. The loop of the plexus dotted with oil, 120 diam. B. The epithelium, containing oil-drops, magnified 360 diam.

plexus is frequently found of a fleshy consistency, probably owing to some interstitial effusion having taken place; and we also find small fatty tumors on the plexuses, as well as a microscopic deposit of fat in the shape of minute oil-globules, dotting the surface of the vessels, but for the most part inclosed in the epithelium.

CHAPTER IX.

THE BRAIN.—GENERAL OBSERVATIONS.

The question that meets us at the threshold of an inquiry into the pathological conditions of the brain, is whether the amount of blood contained within the cranium in the adult, can vary. There is no difficulty in determining the question in the child, for there, as long as the fontanelles are unclosed by bone, the cerebral circulation necessarily obeys exactly the same laws as rule the general circulation; when the skull is completely formed, the pressure of the atmosphere is in a measure withdrawn from the contents, and the variation in the amount of blood contained in the cerebral vessels is very much lessened. Still, in applying the law of atmospheric pressure to this subject, we must remember that the column of blood reaching from the heart to the brain is not like the barometer, a single tube, with a vacuum at the closed end, but that the vessels may be compared to a curved tube, both ends of which are equally under the control of this law. If it were not so, respiration could not, independent of the heart's beat, exert any influence upon the cerebral circulation. The presence of the ventricles, and of serosity in the cavities and subarachnoid fluid of the brain and spinal canal, is a further indication that there is a provision to meet this species of variation—for it is in obedience to this very law of atmospheric pressure, to assume that these fluids mutually assist one another, and as the walls of the cranium cannot collapse, keep up the balance of the circulation by vicarious action. We have already seen that nature shows her horror vacui in senile hydrocephalus, where, if our explanation is correct, the effusion is mainly due to the atrophy of the brain. While we admit, therefore, that the constriction placed upon the vessels of the brain and the peculiar character of the cranial contents, prevents as great a variation in the amount of the fluid contents as takes place in the thoracic or abdominal viscera, we feel assured that a variation does take place, and sufficient to account for many of the phenomena of nutrition and disease. It is important to place this question on a proper basis, as it is one that constantly suggests itself to the pathologist; without a satisfactory explanation, we shall be constantly at a loss to find the proper terms for morbid conditions, and they would themselves appear to contradict our theories. We cannot enter further into this question, but we hope that the suggestions just thrown out may serve to reconcile some conflicting views on the subject. The positive denial of various physiologists, of any variation in the quantity of blood in the brain, apparently supported by such experiments as those of Dr. Kellie,

has served not a little to confuse the student. Dr. Burrows[1] has investigated this question fully, and from experiments and physiological considerations, arrives at the conclusion, which seems unavoidable to the practitioner of medicine, that the quantity of the blood within the cranium is extremely variable at different times, and under different circumstances. There is a peculiar feature in the white matter of the brain, which constantly forces itself on the notice of the microscopist, by impeding his investigation, and which has a strong bearing on the matter—it is the great elasticity of the medullary tissue; this resiliency, among others, is a counterpoise to the rigid structures enveloping the brain. All inquirers are agreed that the relative amount of blood in the different sets of vessels, in the veins and arteries, varies considerably; and on this point Dr. Kellie's experiments appear to afford conclusive evidence, though the examination of a few bodies would suffice to show the same thing.

The importance of the study of the morbid phenomena met with in the brain is self-evident, both from the rank of the organ in the economy, and from the great frequency of cerebral disease. Thus we find, on referring to the Registrar General's valuable statistical reports, that the deaths caused by diseases of the brain rank fourth in order of fatality. The average percentage of mortality in London from zymotic, tubercular, pulmonary, and cerebral disease, appears from analysis of the deaths in ten weeks, selected promiscuously throughout the year 1851, to be respectively 19.9, 18.3, 15.5, and 12.1.

We frequently make post-mortem investigations in cases where all the symptoms indicated that death proceeded from cerebral lesion, and where, nevertheless, we are unable to discover any disorganization such as would appear to justify the conclusion that this was the case. A certain amount of hyperæmia in some of the cranial contents may be all that presents itself to us, and even this may be absent. Sir Astley Cooper's experiments[2] upon rabbits, in which the vertebral arteries were alternately compressed and relaxed, after the carotids had been previously tied, have sufficiently demonstrated the influence of the circulation upon the functions of the brain. The compression invariably produced an instant arrest of respiration, convulsions, and apparent death, and when the finger was removed from the artery the animal gradually recovered. The symptoms closely resembled those of epilepsy in the human subject, to the illustration of which disease they are frequently applied. We have yet to determine the ratio in which mere pressure influences the cerebral functions, as compared with the frequency in which disturbance is excited, and a fatal issue produced by a poisoned condition of the blood, as in uræmia, resulting from granular degeneration of the kidneys.

[1] On Disorders of the Cerebral Circulation, London, 1846.
[2] Guy's Hospital Reports, vol. i. 1836, p. 465.

CONGESTION.

When the congestion of the brain is considerable, the entire organ may present an increase of volume and turgidity. On slicing it, the gray matter may exhibit a deeper tinge than usual, but its natural hue prevents the alteration of color from being very perceptible. The white matter shows an increase of the red dots indicating the bloodvessels, and may, as it does particularly in children, assume a general pinky tint from the same cause. This must not, however, be confounded with the color imparted to it by the knife as it divides the bloodvessels, and, according to the amount of blood contained in them, smears it over the brain surface; by carefully wiping or scraping it, we shall be enabled to determine to which cause the color is due. Scipion Pinel[1] dwells very forcibly upon the occurrence of congestion of the gray matter of the brain as the main pathological feature accompanying mania. He describes the inner layer of the cortical tissue as presenting a lively red or violet tint, the white matter being less altered in this respect, but also offering a livid hue, with occasional blackish patches, or more or less extended ecchymoses. Congestion of the meninges is more frequently found independently of congestion of the brain than the converse; but we must look for the latter more especially in cases in which death has taken place in consequence of poisoning from opium, in epilepsy and apoplexy, in bronchitis, hooping-cough, in fever accompanied by coma, and in hypertrophy of the heart and granular kidney. In a therapeutic point of view, the relation of congestion to cerebral symptoms is important, and the proceeding of Mr. Parry,[2] to compress the carotids in epileptiform and other affections, probably dependent upon this derangement, is a practical application of the doctrine to therapeutics. The pathological effect of arresting the circulation in the vessels of the head is illustrated by cases like those referred to by Dr. Abercrombie,[3] in which, accidentally or intentionally, these vessels were closed, and animation temporarily suspended, until the constriction was removed. The frequent fatality of apoplectic affections, without leaving any appreciable trace, has been the cause, as the same author most justly observes, of the number and variety of speculations on the subject, which have certainly not tended to clear up the difficulty. Turgidity of the bloodvessels in the membranes and brain has been observed in the majority of lunatics, independent of other lesions; thus, Dr. Webster, in analyzing the records of Bethlem Hospital, finds this the case in eighty-nine out of one hundred and eight.

Local congestions are occasionally met with limited to individual portions of the encephalon. There can be no doubt that an anæmic condition of the brain, as well as a cachectic state of the blood circulating in it, may induce, in a similar manner, disease and a fatal issue without offering any perceptible lesions. In these cases, the fibrous tissue of the

[1] Traité de Pathologie Cérébrale, p. 193. Paris, 1844.
[2] Collections from the Unpublished Writings of the late C. H. Parry, 1825.
[3] Diseases of the Brain, &c., p. 211. Edin. 1845.

brain presents a more deadly white, and fewer red spots than in the normal condition, but we possess no means as yet of determining these relations by actual measurement; and in many instances where no organic change has taken place, the tonicity of the arteries may, in articulo mortis, restore the balance of the circulation in such a manner as really to remove all post-mortem effects.

HEMORRHAGE.

Congestion is a transition state to numerous cerebral lesions which leave sufficiently perceptible post-mortem effects. The first of these that we shall consider is hemorrhage, the most frequent cause of cerebral apoplexy, and an affection peculiarly belonging to advanced life; the disposition to it increasing in a direct ratio with the years of the individual. The greatest fatality, according to Dr. Burrows, exists between the age of sixty and seventy, while it is also found to occur more frequently in males than in females. The following table, which has been compiled by Dr. Burrows, clearly exhibits the progressive ratio of apoplexy with advancing age.

OBSERVERS.	20 to 30 yrs.	30 to 40.	40 to 50.	50 to 60.	60 to 70.	70 to 80.	Above 80.	Total.
Dr. Abercrombie	3	4	6	7	7	1	0	28
Dr. Bright	4	4	8	4	5	1	0	25
Dr. Andral	3	3	4	6	5	1	0	26
Dr. Rochoux	2	8	7	10	23	12	1	63
Dr. Hope	2	2	9	6	7	11	2	39
Dr. Burrows	2	9	6	8	7	1	1	34
Total in periods of 10 years,	16	30	40	41	54	30	4	215

This corresponds in the main with the results obtained from the Registrar General's reports. To explain the apparent diminution after the age of seventy, we must remember that the total number of living is very much reduced, and that hence the relative number of apoplectic cases is probably even larger than at an earlier age.

The amount of hemorrhage varies from a spot of the size of a pin's head to an accumulation of many ounces; the former, which may be termed capillary hemorrhage, is frequently observed in connection with effusions of a more extensive character, but may often be assumed to have occurred during life, where trifling apoplectic symptoms have rapidly passed off under appropriate treatment. It is not generally easy to trace the vessels from which the blood has been effused, and there is also much obscurity as to the actual nature of the morbid action which induces the hemorrhage. All parts of the encephalon may present apoplectic effusions, but the parts most frequently affected are the anterior lobes, and especially the vicinity of the corpora striata. As a rule, the gray structures, including the convolutions of the brain, ex-

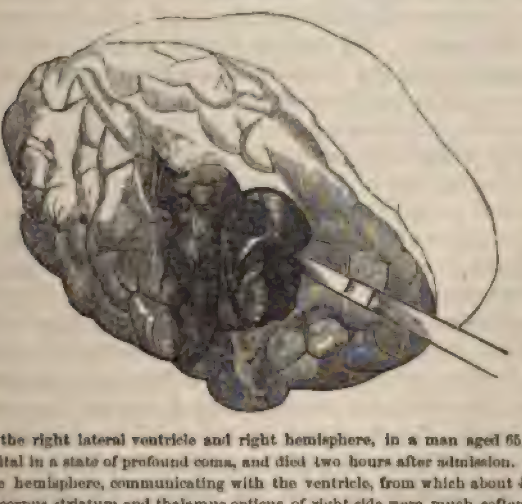
hibit the greatest proclivity to the affection, which is in consonance with the known vascularity of these tissues. The structures most

Fig. 124.

Apoplectic effusion upon the left side of the pons varolii.

removed from the gray matter, as the corpus callosum and the fornix, are least liable to it. Apoplectic effusion is not very frequent in the cerebellum, but it is found to be more rapidly and invariably fatal when it occurs here than elsewhere. Andral's extensive sphere of observation has only presented him with six cases of hemorrhage into the cerebellum, and in three of these it was associated with hemorrhage into the cerebrum. In the causation of apoplectic effusion three elements come into consideration; the condition of the blood, the state of

Fig. 125.



Hemorrhage into the right lateral ventricle and right hemisphere, in a man aged 65. He was brought into St. Mary's Hospital in a state of profound coma, and died two hours after admission. There was a large ragged cavity in the hemisphere, communicating with the ventricle, from which about 4 oz. of black fluid blood escaped. The corpus striatum and thalamus opticus of right side were much softened. There was no apparent disease of the arteries.

the coats of the vessels and that of the tissues surrounding them. The first, though the most important, as necessarily influencing most mate-

rially the two latter, we do not as yet possess sufficient data to speak otherwise than hypothetically of. It is not difficult to conceive that stasis in individual vessels, the formation within them of exudation matter, or an alteration in the density of the liquid, may mechanically and vitally influence the origin of this morbid state. The fact of the occurrence of sanguineous effusion, as a sequel of renal or cardiac disease where no disorganization of the brain is traceable, is a further point corroborative of the position. We may also mention that Messrs. Andral and Gevarral are of opinion that an essential connection exists between cerebral hemorrhages and a diminution in the amount of fibrin in the blood with an increase in the quantity of blood-globules. In eight venesections, performed upon seven apoplectic subjects, they found the fibrin below the normal standard in five, and the globules above it in four cases. With regard to the coats of the vessels, the influence of chronic arteritis in causing pulpy softening or in giving rise to calcareous or atheromatous deposits, and thus rendering them unfit to bear the pressure of the blood-current, is a subject of frequent observation, though disease of the bloodvessels is not a necessary accompaniment of apoplectic effusion. In the same way as this pathological condition induces local affections in the thoracic and abdominal viscera, it causes aneurisms or ossifications of the arteries of the brain, and in both instances affects nutrition, and is liable to be followed by rupture and the effusion of blood. Such alteration in the nutrition of the nerve-tissue as diminishes its resistance to the impulse it receives from the sanguineous current, is the third element to which we have alluded. We shall consider the main characters of softening more in detail further on; but it is necessary to mention here that it occurs in two forms, which appear to be essentially different, the one being due to a state of hypersthenia or active inflammation, the other to an impaired or cachectic nutrition of the tissue. We cannot always in the dead subject demonstrate the sequence or the relation which these conditions bear to one another; but an extended knowledge of morbid processes elucidated by vital dynamics, the microscope, and animal chemistry, will undoubtedly clear up many difficulties connected with cerebral disease, as it has already done in diseases of other organs.

In the present state of our knowledge, and of physical examination, the proximate causes of cerebral apoplexy frequently elude the inquirer; but it is difficult to assume that it can occur without previous disease within the cranium of the nerve-matter, as well as of the vessels; the fact that apoplectic effusion is not met with in healthy individuals who have died from strangulation, alone affords sufficient evidence that a mere arrest of the current of the blood is incapable to produce it; for in these cases the vertebral arteries may fairly be assumed to convey blood to the brain after its return by the veins has been arrested.

The apoplectic effusion is not necessarily fatal in proportion to the amount of blood discharged from the vessels; but the rapidity of the issue appears to bear a relation to the vicinity of the hemorrhage to the medulla oblongata. The effusion of blood into the ventricles is also marked by being very rapidly fatal. Much, probably, also depends upon the amount of laceration of the cerebral tissue accompanying the hemor-

rhage, inasmuch as the curability of apoplectic effusions seems to be in the ratio of the interstitial character of the discharge. Thus, Messrs. Foville and Ollivier have pointed out, both with regard to the encephalon and the spinal cord, that the cure of paralysis resulting from rupture of the nerve-tissue is never complete; but that where the patient is restored to perfect health, the hemorrhage has mostly separated and compressed the cerebral or spinal fibres.

The processes that occur in the blood itself, after it has been effused within the brain, are: the formation of a coagulum, the gradual absorption of the fluid parts of the blood, the formation of an organized membrane around the clot, and the continued absorption of the latter. The rapidity with which these changes occur differs considerably, and depends greatly upon the healthy condition of the surrounding parts. Thus, while Dr. Macintyre[1] has recorded a case of apoplexy, in which, thirteen days after the seizure, the cyst was found fully formed, organized, and nearly empty, a French physician, Moulin,[2] mentions one of seventeen years' duration, in which a cyst was found containing four ounces of sanguineous fluid. When the effusion occurs in the cavity of the arachnoid, we have seen that cysts also form, but we do not meet with them in the cavity of the ventricles, though there is reason to suppose that the blood may be absorbed from their surface also. According to the degree of absorption, the clot changes its consistency and color. The clot first assumes a deeper color and becomes of a chocolate hue, and, from absorption of the serum, is rendered hard; the coloring matter is more and more absorbed, a light-colored fibrinous mass is then seen, much contracted from the original dimensions of the clot, and finally this too may disappear, leaving no remains of the hemorrhagic effusion but the contracted empty cyst, the walls of which are frequently connected by fibrous beads. The cyst itself, in its turn, shrinks up, and finally nothing may remain but a cicatrix. The hæmatoid crystals, first discovered by Sir Everard Home, and more recently described by Virchow, are occasionally found in apoplectic clots, together with orange-colored granular matter. Virchow[3] states that the earliest period at which he has discovered them was seventeen days after the injury; their not occurring in recent effusions has been urged as a positive proof regarding the age of a clot.

It is manifest that no effusion can take place into the cerebral tissue without a certain amount of disruption of the nerve-matter, portions of which may be generally traced within the fresh clot; the greater the previous cerebral softening, the more we shall find the brain comminuted. The danger to life, as the recorded cases teach us, is in proportion to their cerebral disorganization; for the tax made upon the powers of the constitution, to repair the injury done, is necessarily greater, the more inflammatory reaction is set up; for, though the clot possesses in itself a tendency to form a cyst without inflammation, and thus aids in the process of reparation, this cannot suffice to repair the injury done to the

[1] Report of Pathol. Society, 1847, p. 11.

[2] Traité de l'Apoplexie, ou Hémorrhagie Cérébrale, &c. Paris, 1819.

[3] See an article on Blood-Crystals, by Dr. Sieveking, in the British and Foreign Medico-Chirurgical Review, Oct. 1853.

cerebral tissue. After the absorption has come to a stand-still, the cyst or the cicatrix may be borne for years without exciting any new symptoms; and thus, if the individual has suffered from a repetition of apoplectic attacks, we shall find one or more such residuary appearances, in a state indicative of the period from which they date. As we occasionally meet with a recent apoplectic effusion that has been effected near the surface of the brain, marked by fluctuation, we find, after the absorption of the fluid, a slight depression in the superincumbent nerve-tissue, or a supplementary effusion of serum into the ventricles.

WHITE SOFTENING—ŒDEMA.

The converse of the condition which we have just been considering, anæmia, is undoubtedly one of considerable importance, and one that may be assumed to be the cause of numerous morbid conditions; but we have a still greater difficulty of determining its existence than that of congestion of the brain. Where it exists, the brain presents a generally pallid appearance, and especially the white matter is remarkably deficient in red spots, and more dead-white than normally. It is an important element in the diseases termed white softening and œdema of the brain, which, therefore, especially as in their turn they may give rise to apoplectic effusion, may appropriately be treated of in this place.

In estimating the degree of white softening, it is important to bear in mind the physiological variations in the density of the brain, according to the age of the individual.[1] It is naturally very soft in infancy, and progressively becomes firmer with the advance of years; and, in old age, as we find a tendency to rigidity of the soft tissues, and to ossification of the cartilages, we discover the brain to present the physiological extreme of density and toughness. The diminished density of a portion of, or the entire brain, constitutes the disease in question; at times, it appears to be due to an increased infiltration of serum, in the cerebral tissue, in which case it may be considered as identical with œdema. It is common in children, complicated with hydrocephalus, or as a product of arrested or perverted nutrition, or exanthematic fevers; the affected portions of the brain often being entirely diffluent. In adults, it is found in connection with phthisis.

White softening is characterized by a loss of cohesion, varying in degree, and is chiefly met with in the parts most remote from the gray matter, as one would expect, if the etiology, as given above, is correct, because they are provided with fewer bloodvessels. Rostan, who was the first to draw attention to cerebral softening, which has since been extensively studied by pathologists, admitted the presence of an inflammatory and a non-inflammatory form; but we owe to Gluge and Bennett the means of discriminating the two with physical accuracy, inasmuch as they have shown, that an essential feature in inflammatory affections of the brain is demonstrated by the microscope. The in-

[1] See a paper by Dr. Sankey on the Specific Gravity of the Brain, in the British and Foreign Medico-Chirurgical Review, Jan. 1853.

flammation corpuscles or cells, produced by the disintegrating changes which accompany the phlogistic process, are invariably found in the cerebral tissues, where there is inflammation; and it is characteristic of the non-inflammatory softening, and of œdema of the brain, that these corpuscles are not met with. The microscope only exhibits the mere debris of nerve-tissue, in a state of greater or less destruction, without any trace of new formations or products. The entire absence, then, of inflammation corpuscles in a softened portion of brain, serves to confirm the opinion of its non-inflammatory character. According to Dr. Todd,[1] white softening is characterized by organic globules, or large cells, containing oily matter, from which he infers that the disease is accompanied by an active process during life, although he regards it essentially as an atrophic condition. Abercrombie and some other writers attribute the form of softening just described, under the term of cerebral softening, to inflammation; an analysis of the cases given by Abercrombie himself, will be found to support the view of the existence of white softening without the physical signs of inflammation. The microscopic discovery of the product of inflammation will, in future, aid the pathologist in determining the question in individual cases.

Œdema of the brain is a common condition of the organ met with in ataxic conditions, such as typhus, or puerperal disease, in exanthemata, heart disease, and anasarca. It is common in insanity. Pinel describes it as the pathological condition characteristic of stupor, or acute dementia. He states,[2] that if the medullary tissue of an œdematous brain be torn, a peculiar feature presents itself; at the summit of each rent, and at the angle formed by the separation of the fibres, whitish filaments may be perceived with the naked eye, which are nothing but capillaries, strongly injected with serosity; they pass from side to side, and when torn, allow a small quantity of serosity to escape; in the normal state, these capillaries are filled with colored blood. On cutting into a brain thus affected, the tissue is found pallid, and the water drips from it, showing a complete œdematous imbibition. The infiltration causes an enlargement of the brain, and consequent flattening of the convolutions.

INFLAMMATION.

We now return to the examination of one of the sequelæ of congestion, which, in some of its forms and products, constitutes a class of very fatal diseases. Inflammation of the brain occurs in the various forms, and gives rise to the same products that we find in other organs, modified, of course, in their characters by the anatomical relations of the organ. We find it as an acute and as a chronic disease. Acute inflammation of the brain is not frequently met with in the dead-house, in the early stages, nor is idiopathic encephalitis a disease of common

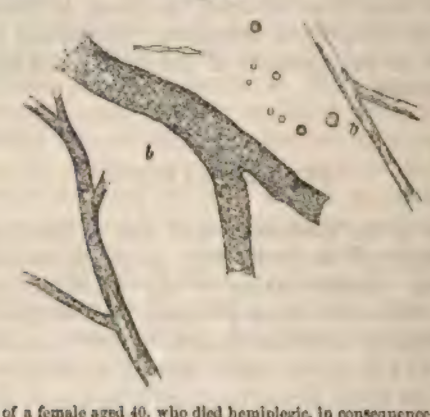
[1] Clinical Lectures on Paralysis, &c., 1854, p. 99.
[2] Traité de Pathologie Cérébrale, p. 257.

occurrence, and many of the cases recorded by older writers resolve themselves into cases of meningitis. It is not our province to inquire into the causes of the peculiarities of diseased action; but we may allude to the circumstance of the brain being so completely withdrawn from physical influences acting immediately upon it, as one of the reasons why idiopathic inflammation should not set up in it, with the same frequency and violence that it exhibits in organs that are more exposed. It is brought on by exposure to the sun's rays in hot summer days or in tropical climates, and may be so rapidly fatal as to produce death before the purulent stage has supervened. Other instances of idiopathic encephalitis are, however, met with in the early stage, in which no such direct exciting cause is traceable. In a case of this kind we find a more or less circumscribed dusky redness in the substance of the brain; the spot generally occupies the upper part of the hemispheres, and, on section, drops of blood may ooze out from the divided surface: there is no necessary change in the consistency of the spot, though it is frequently somewhat softer than the healthy tissue; as the disease advances, the exudation of lymph and suppuration ensue, and the color and consistency of the affected part are modified in proportion. These products are much more frequently the result of chronic and of secondary, than of acute and primary, encephalitis. So much so, in fact, that softening, which is the most common effect of inflammatory action, has been treated, by many authors, as an idiopathic disease.

INFLAMMATORY SOFTENING.

The consistency of cerebral tissue may be altered by inflammatory exudation alone, or with the supervention of suppuration. In the

Fig. 126.



Vessels from the brain of a female aged 40, who died hemiplegic, in consequence of red softening of the right hemisphere.

former case, we shall find traces of congestion, giving to the affected part a reddish hue; and in the softened tissues, the microscope will

show, besides broken-down nerve-matter, a large number of exudation corpuscles; while, as soon as suppuration has occurred, there is a change of color, and we may expect to find pus-cells in addition to the former. Exudation matter may, however, be found where there is no perceptible change in the consistency of the tissues, or other palpable lesion, and it is here that we feel the great advantage of the microscope in assisting our views on the pathology of disease, because it often serves to account for symptoms that otherwise are not to be explained. The vessels, especially, appear covered with, or to contain, the molecules of exudation matter, without the presence of the exudation corpuscles and masses, which are evidently a further stage in the exudation process. The absence of pus-cells would not be an absolute proof that suppuration had not occurred; for they are not always present[1] in undeniable abscesses, where molecular granules and pyoid bodies may be the sole objects detected by the microscope. We are further borne out in this view by Dr. Bright,[2] who expressly states that we can hardly restrict the term, abscess of the brain, to those very rare cases in which well-formed pus is found in the substance of the cerebral mass.

Suppuration occurs in three forms: we find it occupying the convolutions in the shape of a ragged ulcer, varying in size from a fourpenny piece, or less, to that of half a crown; or the pus is infiltrated through a large extent of cerebral tissue, causing what Dr. Bright terms the diffused abscess; or again, the pus becomes limited, as in other parts of the body, by a membranous expansion, and we then have to deal with the encysted abscess. The consistence and color of the parts in which exudation and suppuration have been effected, vary according to the extent to which the tissues are involved; the softening may be scarcely sufficient to mark a difference between the healthy and the diseased portions, and it may reach such a degree that the latter are perfectly diffluent; the discoloration, in the same way, will be more or less straw-colored or reddened, according to the amount of suppuration, the injection of the bloodvessels, and the accompanying exhalation of blood, or its coloring matter. We have already alluded to the presence of exudation corpuscles, which are detected by the microscope in inflammatory affections of the encephalon. They will here be found in large numbers, and will assist in establishing our post-mortem diagnosis, in addition to the evidence, afforded by the microscope, of the presence of true pus-corpuscles. The necessary disorganization of the cerebral tissue, which must ensue in each of these cases, causes the presence of broken-up nerve-matter, which the microscopic specimens will be found to contain. When we have to do with red or apoplectic softening in which hemorrhage has occurred, we shall also detect blood-corpuscles in a more or less altered state. A rough way to determine the presence of softening is, to allow a gentle stream of water to fall upon the suspected part; the softened parts will more or less readily give way and break up.

[1] Lebert, Physiologie Pathologique, vol. ii. p. 303.

[2] Reports, p. 171. See also Bennett on Inflammation of the Nervous Centres, Edinb. Med. and Surg. Journal, 1842–43.

With regard to the locality of red or inflammatory softening, authors are not agreed as to the preponderance of its occurrence in the gray or the white matter; while Gluge and Durand-Fardel are of opinion that it is more frequently met with in the former, Dr. Bennett's researches lead him to assert, that the white matter is the chief seat of this morbid action. French physicians look upon softening of the superficial laminæ of the convolutions as peculiar to dementia, and state that it is characterized by portions of the gray matter adhering to the meninges when they are removed.

It follows, from what we have stated, that some of the distinctions which authors have laid down with regard to the varieties of softening, as characterized by their color, are not essential differences, but rather, different stages of the same inflammatory process. The structure of nerve-matter would, *à priori*, lead us to expect that the products of inflammation would assume an appearance different from what they present in other tissues of the body, and as we advance in our knowledge of cerebral disease, we shall probably succeed in reducing it still more to the general type of morbid action. Dr. Abercrombie has suggested that ramollissement of the cerebral substance is analogous to gangrene, occurring in other parts of the body; but, though obliteration of the small arteries of the brain may give rise to this change, it is by no means an essential cause; and we may sum up the prevailing views on the subject, in Dr. Bright's classification of the *causa proxima* of softening; he attributes it—(1) to an impediment in the circulation; (2) to congestion; and (3) to inflammatory action. The reader will gather from our remarks, that we do not concur in the view of Dr. Abercrombie; an additional argument against it is, that aneurism of the cerebral arteries is not commonly followed by softening; and, if anywhere, we should expect to find it in this case most uniformly, if the process is identical with death of the part. Those cases of ramollissement which we have attributed to mal-nutrition, or a cachectic state of the blood, as opposed to those resulting from inflammation, would be more appropriately classed under the head of gangrene; and under this head may also be placed the cases of softening resulting from ligature of the carotid artery; but we are not justified in assuming the presence of gangrene in parts which, though seriously diseased, are by no means withdrawn from the range of vital processes. An important physical symptom of gangrene is remarkably absent in softening of the brain, viz: a fetid odor.

Softening of the brain is looked upon as an essentially fatal disease, though there is no inherent reason why the process of resolution and absorption should not be carried out within the brain as in other organs. It is probable that, as our means of diagnosis become more perfect and more refined, our knowledge of this subject will also enlarge; at present, we deal rather with the last stages of the disease than with its incipient and more curable features. One reason why inflammatory softening appears less tractable than analogous processes elsewhere, is that the brain does not possess a great power of isolating the disease; encysted abscess is a very unfrequent occurrence in cerebral pathology. The greater number of cysts containing pus, that we meet with, are the

result of external injury, involving the bone, as if nature only cared to protect the brain from contact with the external atmosphere. It was already observed by Dr. Baillie, that abscess on the surface of the brain was almost constantly the effect of external violence, but that it was often independent of this cause when formed at a considerable depth within the brain, and that the former was by far the more common form. The cyst itself presents in either case various degrees of thickness and density; the former may amount to half a line, and the latter increase to the consistency of leather. The cysts themselves have a laminated fibrous structure, and they are lined with a layer of the pyogenic membrane.

With regard to the cause of softening, as of cerebral disease generally, it must be mainly sought for in changes directly and primarily affecting the brain; but that peripheral affections may be followed by central disease, is forcibly illustrated by the case published by Lallemand[1] of a soldier, who had been operated upon for aneurism of the right axillary artery. In applying the ligature, the nerve was inclosed, cerebral symptoms followed on the seventh day, and death ensued on the eighth: the post mortem showed an abscess in the left posterior cerebral lobe. The case is also of interest, as affording proof of the uniformity of the law of crucial conduction.

In phlebitis and purulent infiltration, which are commonly associated with cerebral symptoms, we do not generally discover any marked trace of disease in the brain. Metastatic abscesses appear limited to the great organs of depuration; but while we rarely, if ever, discover metastatic abscess in the brain, in consequence of phlebitis in other parts of the body, it is not uncommon to find metastatic abscess in the liver or spleen, after idiopathic phlebitis in the veins of the brain.

INDURATION—HYPERTROPHY.

The converse of ramollissement or induration, appears, like the former, to be an occasional result of a phlogistic process; but, like it, we must in many instances attribute it to a non-inflammatory change in the nutrient sphere. We find portions of the brain both at the surface and in the deeper-seated parts presenting no material alteration beyond an increased density as compared with surrounding parts. This is distinct from the hardened cicatrices resulting from the absorption of apoplectic effusions. A general hardening of the brain, accompanied by a livid earthy hue, and an increase of the entire volume of the organ, is met with in chronic lead poisoning;[2] the convolutions are found flattened, the ventricles are compressed, the tissue is dry; and a chemical analysis will detect the presence of sulphate of lead in the brain. Tanquerel des Planches records two cases in which this was done. It is also found in cases of acute lead poisoning.[3] A similar state of in-

[1] Recherches Anatomico-pathologiques sur l'Encéphale, &c., vol. i. 123.
[2] Tanquerel des Planches, Traité des Maladies de Plomb, Paris, 1839, vol. ii. p. 298.
[3] Alfred Taylor on Poisons, &c., p. 133.

duration and hypertrophy of the white substance, is stated by Ferrus and Parchappe to be found in epilepsy. Laënnec and others have also directed attention to hypertrophy as simulating hydrocephalus. We must also be careful not to confound cases of tumefaction of the brain from softening with hypertrophy; thus we find, in Andral's *Clinique Médicale*, the account of a post-mortem, in which, owing to this cause, the left hemisphere was so much swollen as to push over the mesial line to the left side. A similar instance is preserved in the museum of St. Thomas's Hospital.

In a case of hypertrophy, on removing the skull-cap, the brain seems to expand, as if it had been previously confined in too narrow a space; the membranes are thin, the convolutions are flattened by being compressed against the bone, and the ventricles are found to contain very little or no fluid. On making a horizontal section, the gray matter is not seen altered, and the naked-eye view displays an increase in the amount of white matter; this, according to Rokitansky, is owing, not to an augmentation in the number of nerve-tubes, or their dimensions, but in the excessive accumulation of the intervening and nucleated substance. We have not ourselves been able to confirm this statement, nor have we found corroborative evidence in other writers. Hypertrophy appears to be due to a lymphatic constitution, and it is met with chiefly in early childhood. As long as the fontanelles are not closed, it does not in itself involve danger, and even the intellect continues unimpaired; but as soon as the fontanelles have closed, the undue pressure gives rise to numerous cerebral symptoms, none of which, however, are characteristic of this disease. It belongs essentially to the family of scrofulous affections; the frequent coincident distortion of the bones, the swelling of the lymphatic glands, and the general torpor of the system, suffice to establish its relationship. The bloodlessness and dryness of the tissues must assist us in deciding, in a doubtful case, whether we have to deal with hypertrophy of this character, or with hyperæmia, hydrocephalus, or œdema, conditions which also may cause the brain to appear too large for its case, and produce a flattening of the convolutions. Nor would it be just to consider as diseased a brain, which, though larger than the average at the age of the individual, exhibits no morbid relations such as those described.

Individual parts of the cerebrum are very rarely found hypertrophied by themselves. Dr. Mauthner[1] records a case of hypertrophy of the thalamus opticus of the right hemisphere in a child of three years of age, which, till within three weeks of its death, had enjoyed sound health. She then fell from her chair, striking the occiput, and became paralyzed on the left side. Shortly before death scarlet fever supervened, she became delirious, and died comatose. The thalamus is described as enlarged to the size of a hen's egg, of a lardaceous, dead-white appearance on section, without softening of the adjacent parts or effusion; except that the testes were very vascular and the left optic nerve enlarged, no abnormity was discovered. Dr. Mauthner is of

[1] Die Krankheiten, des Gehirns, und Rückenmarks, bei Kindern, von L. W. Mauthner. Wien, 1844, p. 189.

opinion that the organic malady had remained latent, until the occurrence of the fall. The same author gives an interesting table, showing the weights of the brains of fifty children aged fifty months and under, who died of various diseases; he concludes that all inflammatory affections have a tendency to increase its weight, and that this increase is mainly due to the greater amount of blood contained in the cerebral vessels.

ATROPHY.

The converse of hypertrophy is a condition which, as we have already had occasion to remark, is the result of a natural process in old age; but it is also the effect of disease; or of an arrest of development. In consequence of long-standing, exhausting illness in children, Dr. West informs us that the brain is found far from filling up the cavity of the skull, so that a knife may be passed, in many places, between it and the cranial walls. The same appearance is met with in the adult, and the consequence is that serum is effused between the brain and its envelops, in order to supply the defect; the convolutions become thinner, and they are separated by broader sulci. There is some discoloration of the tissue, and the veins of the pia mater are observed to present a varicose appearance, owing to the loss of the support which they experience. We also meet with partial atrophy; this is generally of a secondary character, owing to pressure exerted upon individual parts by tumors or other adventitious growths, apoplectic cysts in the arachnoid or peripheral lesions. The absorption induced by the effect of pressure is accompanied by induration of the adjoining layers of the cerebral tissue. The atrophy that is found as a result of arrest in the functions of a peripheral nerve, as in the case of the optic thalamus in amaurosis, is a marked instance of the influence of functional derangement upon nutrition, propagated to a distance.

In idiots, we have congenital atrophy of the entire, or of portions of the brain; in the brain of old insane persons it is common, according to Neumann's statement, who has examined fifty cases of the kind, to find the posterior lobes and their convolutions more atrophied than other parts; Sir Charles Bell, Cruveilhier, and Lallemand record cases of epilepsy and hemiplegia, accompanying congenital atrophy of one hemisphere, though not necessarily associated with a destruction of the intellectual powers.

CHAPTER X.

THE BRAIN—MORBID GROWTHS.

The products of a perverted state of nutrition and of a cacoplastic condition of the blood, which are found in other tissues of the body, also occur in the brain, though for the most part in a secondary form; associated with or following upon their deposit elsewhere. They have certain features in common, owing to the anatomical relations of the brain; thus, they all generally assume a rounded shape, owing to the uniform pressure to which they are subjected on all sides; they are not, as for instance in the lungs, received into a mould by which their external configuration is in a measure determined; they do not proceed to a similar degree of development from the early danger to life which they induce; they are liable to produce softening of the tissues in the immediate vicinity, and, owing to the impairment of the circulation, are commonly accompanied by an effusion into the ventricles. The last two circumstances are those to which we may probably refer the symptoms observed during life; for the presence of tumors in the brain is often not discovered until the death of the patient from disease of some other organ, and where they had maintained this quiescent state, the cerebral tissue in the vicinity exhibits no traces of degeneration. Thus, Messrs. Tonnellé,[1] Léveillé, and others, who have devoted especial attention to the occurrence of cerebral tubercle, conclude that the tubercles in themselves do not give rise to any symptoms, but that the cerebral symptoms accompanying them are exclusively due to the intercurrent inflammation. The difficulty of early diagnosis, and the fact of adventitious growths in the brain occurring almost exclusively in the secondary form, place them more especially in the range of the morbid anatomist; they are even less amenable to therapeutic treatment than when they have found a nidus in the abdominal or thoracic organs. The form in which we most frequently meet with them is tubercle and cancer.

TUBERCLE.

Tubercle, as we have already had occasion to see, is a frequent concomitant or source of meningeal inflammation. Tubercular deposit in the cerebral tissue, like the former, is equally a disease peculiar to childhood; but the two are not necessarily associated together. Tuber-

[1] See Rilliet and Barthez, Traité Clinique des Maladies des Enfants, tom. xiii. p. 552, seq.

cle in the brain may affect any part of the organ; it occurs in the shape of rounded nodes varying in size from a pin's head to a walnut or hen's egg; the deposits are not generally numerous, and their size bears an inverse ratio to their frequency. It is most common to find only one or two, and of an average size of a chestnut. Dr. Baly has recorded a case in the reports of the Pathological Society[1] of a young man who died in the Millbank prison, and in whose brain tubercles were found; only two were discovered in the left hemisphere, but the number in the right are estimated to have been as many as fifty, varying in size from a grain of pearl barley to that of a barley-corn; the same case is also instructive as showing the great rapidity with which the deposit may occasionally take place under circumstances favoring the disease; for the patient was admitted into the prison on the 30th December as a healthy subject; after a few days was attacked with headache, and on the 19th of January following, he died with all the symptoms of an acute cerebral affection. The case appears to disprove the dictum of Rokitansky, that cerebral tubercle never occurs in any but the chronic form.

The tubercle presents the appearance and consistency of soft yellow cheese, and while miliary granulations are peculiar to the meningeal form, the yellow tubercle is the variety almost invariably met with in the brain. We are unable to determine whether the deposit takes place in this form; the more extended application of the microscope will best decide the question; since the microscopic characters of tubercle are sufficiently determined to give a positive answer. Rokitansky is of opinion that tubercle in the brain, does, in part at least, commence in the gray translucent form, for portions of a tubercular mass are sometimes found in that state. In any case, however, he adds, it may continue for a short period only in that form, and soon pass into the stage of the yellow cheesy tubercle. The microscopic elements are the same granulated nuclear corpuscles of an ovoid or somewhat irregular shape, interspersed with granular blastema and particles of oily matter found elsewhere; according to the amount of inflammatory action exerted in their vicinity we shall also find more or less glomeruli, with other traces of its effects. The tubercle is generally surrounded by a delicate cyst, and when, which occasionally happens, the tubercular matter proceeds to the stage of softening, the superficial observer may mistake the morbid appearances for those of a simple abscess of the brain. The concurrence of tubercle in other parts, together with the aid of the microscope, will assist in fixing the real character of the affection. The frequency of tubercle of the brain in children is a point not to be forgotten in the treatment of their diseases, and is one of the reasons why the overstraining of the mental faculties of a delicate child is so much to be reprobated. Physicians who have had the most extensive experience in these matters agree as to the rarity of its occurrence in the adult; thus, Cruveilhier never met with a single case, and Lugol, in the large hospital of St. Louis, has only seen eight instances, in none of which any symptoms of the disease were manifested during life.

[1] Session 1850–51, p. 34.

It is a singular fact, which we gather from the statistics of MM. Rilliet and Barthez, that sex appears to exert a marked influence upon the occurrence of cerebral tubercle; in each variety, the males are considerably more liable to the affection than females; of forty-four cases, we find twenty-nine occurring in boys, and fifteen in girls. The fact is confirmed by the statistics of our own medical writers: an analysis of fifteen cases reported by Dr. Abercrombie and others, establishes a similar proportion; ten of these cases were males, and five females. It is, however, right to state that Dr. Hennis Green's[1] statistics contradict this fact; his observations were made at the same hospital as those of MM. Rilliet and Barthez, and, of the thirty cases which he has collected, fourteen occurred in boys and sixteen in girls.

The deposit of tubercular matter sometimes occurs in patches of irregular shape and size, on the surface of the brain, beneath the pia mater,[2] but commonly, as we have already seen, it forms nodules within the cerebral tissue. It is often met with both in cerebrum and cerebellum at the same time; the number of cases in which it occurs in one or the other alone is about equal; the pons varolii is, in rare cases, the only seat of the deposit.

In the lungs, we frequently meet with satisfactory evidence of the power of the system to reject and cure tubercle; we are not possessed of similar proof with regard to the brain; the only analogous process is that in which the vitality of the deposit seems utterly destroyed, and cretification results; this is a metamorphosis which sometimes, though rarely, takes place in cerebral tubercle. We must not confound with cretification of tubercle, certain gritty or sabulous masses found in the brain; thus, our notes contain the history of a case, in which, on a vertical section of the cerebellum, the knife grated upon some calculous formations, imbedded in the tissue, and intimately adherent to it; there proved to be, on each side, three or four irregularly crystallized masses, which broke up easily on pressure, and were not affected by either liquor potassæ or acetic acid. There was no other perceptible disease of the cerebral tissue, but the choroid plexuses were covered with concentric corpuscles. Andral[3] gives, as a great curiosity, an analogous case, in which, however, the "ossifications" were inclosed in a cyst.

CANCER

Next in frequency to tubercular deposit we find the various forms of cancer, all of which, excepting the epithelial variety, are met with in the brain. There are no symptoms peculiar to the disease, beyond the effects resulting from pressure; and even they do not appear to be in any way commensurate with the size of the deposit. The form which it assumes is that of infiltration, without any definite limits, or of a tumor surrounded by a cyst; in the former case, there seems a continuity of tissue between the cancerous mass and the nerve-tissue. The

[1] Medico-Chir. Trans. vol. xxv. p. 192.
[2] See Mr. Dunn's case, *ibid.* p. 209.
[3] Clinique Médicale, t. v. 719.

same law, with regard to the predominant liability of the cerebrum compared with the cerebellum, applies in the present instance; thus, in forty-three cases of cancer of the nervous centres alluded to by An-

Fig. 127.

Cancerous tumor, occupying the upper portion of the posterior cerebral lobe of a man aged 54, brought into St. Mary's Hospital comatose and hemiplegic, in which state he remained until death. The central portion was dense and fibrous, of a yellow color, and consisting of fusiform fibre-cells: the external portion soft, cream-colored, or pink, composed of a variety of compound cancer-corpuscles. The whole was surrounded by a red vascular margin.

dral,[1] we find thirty-one occurring in the cerebral hemispheres, and five in the cerebellum; the remainder were thus distributed: three were found in the pituitary gland, one in the corpus callosum, and three in the spinal cord. The same author, by an analysis of the cases collected by him, establishes, numerically, the relation commonly found to prevail with regard to the frequency of primary cancer in the nervous centres; in ten of the forty-three cases only, or less than one-fourth, was there any carcinoma in other organs. As a result of local injury, we meet with a species of fungoid growth of the brain, which has been termed hernia cerebri, but which is very different from the hernia cerebri to which allusion has been made, in the section treating of congenital hydrocephalus. In consequence of a fracture of the skull, the brain appears to sprout forth in the shape of a vascular, medullary growth. It appears as if the brain, released from its confinement, luxuriated in its newly-acquired liberty. Extensive suppuration is generally found accompanying this form of hernia, within the brain. We are not in possession of any microscopic examination showing the exact nature of the growth.

In addition to tubercular and cancerous deposits we also meet with melanotic, fatty, and fibrous growths, and cysts in the encephalon.

It was long doubted whether melanosis ever occurred in the brain, and it certainly is rarely found in this locality. Sir Robert Carswell,[2] however, gives a specimen of two tumors of this description, which

[1] Clinique Méd. t. v. p. 688.
[2] Pathological Anatomy, 1838, Art. Melanoma, pl. ii.

were located in the right hemisphere of the cerebrum of a man; they were of the size of a hen's egg, and penetrated into the ventricles. Melanotic deposits were also found in other organs of the same subject, and the veins passing from the tumors in the brain, were observed to contain melanotic matter in a fluid condition. Dr. Hooper's work on the brain also contains a plate representing this disease; and, recently, Dr. Clendining brought a case of the kind before the notice of the Pathological Society.[1]

The simple fatty tumor is only found in the choroid plexus, where it does not, however, attain any great size. The fatty growths most frequently met with in the brain are those which are termed cholesteatoma; they are formed of concentric layers, and present a metallic lustre; they consist of membranous layers, ordinary fat-vesicles, and cholesterin plates, and are inclosed in a capsule. They attain the size of a walnut or goose's egg.

FIBROID TUMORS.

We have searched in vain for the evidence of fibrous tumors occurring in the cerebral tissues, though their existence is admitted by Rokitansky. None of the pathological works that we have consulted record a case of the kind; we are, therefore, inclined to assume that they are limited to the dura mater, and that fibroid tissue only occurs in the metamorphosis of old cysts or envelops of the heterologous growths found in the brain.

CYSTS.

Cysts of various kinds present themselves in the brain; those resulting from apoplectic effusions are the most common, and present, as we have seen, various stages of development. It is probable that, in many instances, their formation may be due to the same process as that described by Mr. Prescott Hewett, as giving rise to the inter-arachnoid cysts, viz: a formation of a false membrane, subsequent to the effusion from the sanguineous clot. The firmness and thickness of these cysts will serve to distinguish them, even when they only contain serum, from the true hydated cyst. The microscope, by determining the presence of entozoa, or the parallel lamination of the envelop peculiar to hydatid growths, will further assist our diagnosis. The only parasites hitherto discovered in this locality, are the ecchinococcus and the cysticercus cellulosæ, which, however, are not to be viewed as essentially distinct. The acephalocyst occupies the peripheral, more frequently than the central portions of the hemispheres, and is found to present no connection with the surrounding tissues. When met with in the brain, there is generally a coincident development of the same parasites in the liver, a fact first pointed out by Aran,[2] who has analyzed forty-seven cases of this kind.

[1] Report of the Pathological Society of London, 1847, p. 15.
[2] Schmidt's Jahrbücher der Medicin, vol. xxxiii. p. 136.

THE PITUITARY BODY.

The pituitary body presents morbid conditions, which, generally, are rather pathological curiosities, than that they offer any peculiar points of general interest; in a physiological point of view, tumors or cysts occurring in it attract attention, from their not producing those symptoms which are generally attributed to pressure upon the encephalon, and this is supposed to be due to the force acting in an upward direction. Thus, they are rarely accompanied by paralysis, though acquiring an extent sufficient to displace the lateral ventricles with the thalami and corpora striata, a circumstance presenting, as Dr. Romberg remarks, an analogy to the different effect produced upon the conduction of a nerve by a tumor, according as the nerve is gradually distended or forcibly compressed. Neither Rokitansky, who treats diseases of the pituitary body in detail, nor Engel, who has written a monograph on the subject, corroborates the observations of Joseph Wenzel, that disease of the pituitary body is an essential feature in epilepsy. The pathologists of our own country have not observed a relation of the kind. Epilepsy is met with as a result of the most various degenerations, or morbid products within the brain, independently of any marked disease of this appendix cerebri; it is not constantly associated with any one lesion, and in the cases of disease of the pituitary gland, given, for instance, by Dr. Bright, we find no epileptic seizures during life; or, conversely, we see epilepsy[1] occurring without any disorganization of this part.

The pituitary body does not seem to bear any definite relation to the manifestations of the mind. Dr. Bright[2] gives an instance in which it was absent; the patient was a man who died at the age of forty-eight, of softening of the left corpus striatum; but he had enjoyed thorough good health until five months previously.

In the course of our account of cerebral morbid anatomy, we have had occasion to allude to the occurrence of aneurism of the cerebral arteries; the subject will meet with a fuller consideration when we treat of the diseases of the vascular system; but we could not dismiss the pathology of the brain without pointedly remarking upon their importance in the production of cerebral symptoms, and as a more or less direct cause of death. The diseases of the arteries play a most important part in the production of cerebral disease, and in many of the morbid conditions which we have passed in review they may be viewed as one of the main elements.

[1] Reports, &c., case cxlii.

[2] An interesting case of disease of the infundibulum and pituitary body is given in the records of the Pathological Society of London for 1849, p. 19.

CHAPTER XI.

THE SPINAL CORD AND ITS MEMBRANES.

We must commence this section with the ungratifying confession that it is a subject upon which our knowledge is very limited. This is partly owing to the various baseless hypotheses which the pathology of the spinal cord has admitted, and still more to the mechanical difficulty which presents itself to the investigation of its derangements, during life, as well as after death. The depth of muscle which invests the spinal column on the dorsal surface, the tediousness of the procedure of sawing through the arches of the vertebræ, and the routine system of conducting post-mortem investigations, militate against the frequent examination of this organ. Nor can we hope that any great amelioration will take place in this respect, until our hospitals and medical schools are able to endow the curatorships of morbid anatomy in such a manner as to secure the undivided services of men of science, for a series of years. So long as the spinal cord was considered only as an aggregation of nerve-fibres, serving to conduct influences to or from the brain, it was natural that its pathological changes should be regarded as of a secondary importance; but since the researches of Dr. Marshall Hall have shown its claim to be considered in the light of a central organ of the nervous system, endowed with powers independent of the brain, the morbid anatomy of the spinal cord has also acquired a higher dignity. As our knowledge of its physiological endowments, and of its structure, is enlarged, and as our means of physical diagnosis are improved, we may hope to see its morbid anatomy better understood and appreciated. At present, we can scarcely be said to have advanced beyond the very threshold of this department of science.

We shall follow the same order we adopted in treating of the morbid anatomy of the brain, and examine successively the post-mortem appearances of the dura mater, the arachnoid, and pia mater, and then of the cord itself. In all cadaveric examinations of the spinal column, it is particularly to be borne in mind that the position of the body after death may influence the post-mortem phenomena, independently of morbid action, owing to the gravitation of the fluids to the depending portions, and their secondary effect upon the nerve-tissues. How important it is to attend to this point, is illustrated by some observations, made by Mr. Curling,[1] of tetanic cases. On examining the body of a man who had died of tetanus, which had been placed on its face immediately after death, Mr. Curling found that part of the pia mater covering the anterior columns of the medulla spinalis remarkably vascular; a circum-

[1] On Tetanus, p. 48.

stance which would necessarily induce a conviction in the mind of the pathologist, that an essential lesion had been discovered, by which the exaltation of motor action could be satisfactorily explained. Unfortunately for the conclusive force of the observation, in three other instances, where the bodies were suffered to remain in the usual position, the vessels on the posterior parts only were observed to be turgid.

This influence of position is more likely to affect the spinal cord than the brain, owing to its being less excluded from atmospheric agency. It is also important to remember that the relation of the envelops of the cord differs from that existing between the investments of the brain and their contents in various material points. The movements of the osseous case of the cord would have rendered a close adhesion with the membranes a source of frequent danger; we may fairly assume this as a reason why the dura mater of the spinal column is only very loosely attached to the vertebral canal; on which account it allows of an accumulation of fluid on its external surface, such as we but rarely meet with in the brain. Owing to the firm attachment of the dura mater to the occipital foramen, fluids accumulated on this part are prevented passing into the cranial cavity, while there is a free communication between the arachnoidal spaces of the two cavities. This fact is one that must not be overlooked in morbid affections both of the spine and the encephalon; independent of the protection that the arachnoidal fluid affords to the cord, it is an evident means of securing a balance in the circulation in the nervous centres, while, on the other hand, a derangement in its quantity and site may be alone sufficient to produce serious symptoms, which the morbid anatomist would be unable to measure by physical tests.

We possess no evidence of the occurrence of idiopathic disease of the spinal dura mater, though it can scarcely be supposed that a fibrous membrane, situated as it is, should not suffer from the rheumatic diathesis. In all cases of injury of the vertebral column it is liable to be affected, and scrofulous disease of the vertebræ and the extension of psoas abscess may involve it. In the congenital affection termed spina bifida, which is analogous to the form of hernia cerebri, occurring in infants as a result of non-closure of the cranial bones, the dura mater extends into the cyst that shows on the dorsal surface of the column; but it is occasionally found deficient at one point, so that the contained fluid is only retained by the thin meninges. This pathological state differs from that to which we have compared it in this, that the fluid is entirely external to the nerve-tissue, and that the cyst at no time presents a layer of medullary matter.

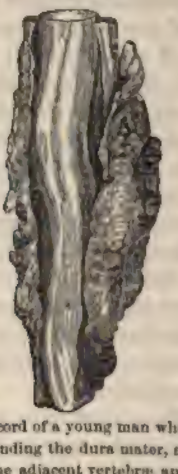
The tumor varies in size from that of a small nut to that of a child's head; it generally is solitary, and occupies the lumbar or sacral region; when occurring in the back we may expect to find another tumor of the same description lower down. The swelling is of a semi-globular, or ovoid shape, and may appear pediculated, owing to a constriction at its base. The thinness of the cutaneous covering passing over the tumor, has induced some pathologists to deny its presence; this however is an error.

The rarity of the occurrence of idiopathic diseases in the dura mater, applies equally to adventitious products. Encephaloid, and other forms of carcinoma, undoubtedly occur primarily in the dura mater, but in

many of the instances on record, it is manifest that the disease extended from the bones to the theca vertebralis. The same is true with regard to tubercular deposits; in both cases, however, the membrane is liable to become secondarily involved, by extension of these diseases either from within or without the canal.

An instance of melanotic growth apparently proceeding from the dura mater of the cord, is to be found in the Report of the Pathological Society for 1847. Dr. Williams discovered it in a patient, aged forty-six, who, three years previously, had suffered from hemiplegia of the right side, following the extirpation of the right eye, for fungus. The patient recovered from this, and in the summer preceding his death was attacked with epilepsy: weakness and numbness of the lower extremities, and inferior portion of the trunk, soon proceeding to complete paraplegia, supervened. The brain and its membranes were found healthy; within the spinal canal, closely adherent to the theca externally, there existed an irregular encephaloid mass, mottled with dark spots, extending from the third to the sixth dorsal vertebra, the bodies of which were carious and infiltrated with cancerous matter; the portion of the cord beneath the tumor was flattened, soft, and wasted. In connection with this subject, we may also be allowed to mention the very rare occurrence of an accumulation of fat, a genuine fatty tumor, within the spinal column, in contact with the dura mater; an instance of this kind was brought before the Pathological Society, in 1852, by Mr. Obré, in which death was produced in an otherwise healthy child by the

Fig. 128.



Part of the dorsal portion of the spinal cord of a young man who died paraplegic. A thick layer of lymph and tuberculous matter was found surrounding the dura mater, and slightly compressing the cord. It was manifestly an extension of disease from the adjacent vertebræ and intervertebral cartilages. The cord and the dura mater appear healthy in texture. From St. Bartholomew's Museum, Series vii. No. 10.

mere mechanical pressure exerted by a deposit of this kind. The lipomatous growth was two and a half inches in length, the breadth of the canal, and about half an inch in thickness, composed of the ordinary spherical fat-cells: it did not differ from fat usually met with in other situations, excepting that the cells seemed to contain fat in a more solid and granular state. It lay between the theca and the bodies of the last cervical and first dorsal vertebræ.

CHAPTER XII.

THE ARACHNOID AND PIA MATER OF THE SPINAL CORD.

ALTHOUGH the anatomical connection between these membranes is somewhat different from that obtaining between the cerebral meninges, it does not appear that their relation in disease differs materially from what we have found to prevail in that locality; we shall, therefore, consider them together.

The absence of valves in the spinal veins, and their peculiar distribution, cause the circulation of the spinal cord to be very sluggish, and, therefore, prone to congestion and stagnation; to this, Ollivier attributes the great number of dilatations which we find in the different points of its extent, in individuals advanced in years. He adds, that he has generally remarked that the quantity of serum in the vertebral canal was so much the greater, according as there was a greater congestion in the veins of the spine, and of the membranous coverings of the cord; thus, the slowness and difficulty of the course of the venous blood may be here the causes of a dropsy, which is independent of inflammation of the spinal membranes. These effusions of serum will, according to the exciting causes, be of a chronic or acute character; in infancy, irritation frequently gives rise to a more rapid accumulation of fluid, while in old age a slow effusion is frequently met with, which Rokitansky attributes, in part, at least, to a secondary congestion, arising from atrophy of the medulla and the roots of the nerves. The fluid exhaled under such circumstances will follow the law of gravitation, and accumulate at the lower end of the spinal cord, and thus assist in exciting and perpetuating paraplegic symptoms of which we may be unable to detect a sufficiently satisfactory reason after death. The occurrence of sanguineous apoplexy of the meninges, in any form, is very rarely met with; Dr. Abercrombie gives a single instance, which occurred under his own observation, in a child, aged seven, in whom, after an illness of three days, death ensued after violent convulsions. A long and very firm coagulum of blood was found, external to the cord, extending the whole length of the cervical portion. An interesting case of hemorrhage under the pia mater, but external to the cord, is also quoted, from Dr. Stroud's notes, by Dr. Bright (p. 340). Numerous instances of spinal apoplexy occurring in children are given by Dr. Mauthner, but as no post-mortem appearances are recorded, the inferences are solely derived from the symptoms, which do not enable us to state positively the exact nature of the effusion. We possess more satisfactory and copious evidence regarding the inflammatory affections of the spinal meninges, and it appears that it is a

very frequent cause of death in new-born infants; thus, Billard found, that in thirty cases of convulsions, there was meningitis of the cord in twenty, only six of which presented inflammation of the cerebral meninges. It is much less frequent in the adult, and is here almost invariably associated with, or consequent upon, cerebral inflammation. As a result of an acute inflammation of the membranes, we find lymph, or pus exuded, to a greater or less extent. Either may invest the entire surface of the cord, or it may be limited, as in a case that fell under our own observation, in a child of four years of age, to a space of an inch and a half in length. But we must be careful in at once concluding that we have to deal with a case of spinal meningitis, because we find the theca vertebralis lined with pus, for it may find its way from without into the cavity; thus, in a case of psoas abscess, given by Dr. Bright, the sudden supervention of fatal symptoms was manifestly due to this cause; a probe could be easily passed from the intervertebral foramina into the adjoining abscess. In the chronic form of spinal meningitis, the traces of the disease consist in greater or less opacity and thickening of the arachnoid, which frequently is found closely adherent to the spinal cord, and corrugated.

The symptoms of spinal arachnitis are met in trismus neonatorum, in the shape of congestion of the spinal arachnoid, with an effusion of blood or serum into its cavity; and Dr West[1] also states that, in the three cases which he examined, he found effusion of fluid or coagulated blood in the cellular tissues surrounding the theca of the cord. This is not necessarily at variance with the statements of Dr Schöller[2] and Dr. Colles,[3] who attribute trismus to inflammation of the umbilical arteries, as this may, and is, found to coexist with the former. Symptoms of inflammation of the spinal membranes have also been met with in tetanus; but in by far the greater number of cases examined after death, no uniform or adequate cause, to which the symptoms were referable, could be discovered. The importance of the predisposing causes is, probably, as great in tetanus as it is shown to be in trismus; atmospheric states have a manifest influence in this respect, and an irritation set up in any part of the body, and propagated to the nervous centres, under such circumstances, induces the disease. The same absence of uniform pathological data exists in another disease, which we cannot but refer to the nervous centres, hydrophobia; congestion of the cerebro-spinal membranes and nerve-matter, and some occasional effusion, is all that is generally met with in the shape of post-mortem effects; we need not add that these appearances cannot be considered as characteristic of the disease in question. Mr. Youatt, whose extensive experience of hydrophobia in the brute creation, justifies our referring to him as an authority, states that the appearance of inflammation of rabies is of a peculiar character in the stomach, but that no conclusion can be drawn from the state of other organs. In discussing the changes occurring in the spinal cord itself, we shall have occasion once more to recur to the subject of tetanus.

[1] The Diseases of Infancy and Childhood, p. 125.

[2] Neue Zeitschrift für Geburtskunde von Busch, D'Outrepont und Ritgen, vol. v, p. 477.

[3] Dublin Hospital Reports, p. 285.

In cases of long standing paralysis and paraplegia, we find evidence of chronic meningitis of the cord in the corrugation, opacity, and close adhesions of the membranes to one another, and to the cord. A good illustration of this is afforded by a case given in Dr. Bright's Reports (page 380); here, the dura mater of the cord was unusually firm and thick, and, as far as the middle of the back, closely adherent to the pia mater, from which, in most parts, it could not be detached without lacerating the cord. On attentive examination, it was found that the apparent thickening of the dura mater depended chiefly on a layer of membrane, of almost cartilaginous thickness, beneath it; and was, probably, rather the diseased arachnoid, or an adventitious deposit, than the dura mater itself. These appearances may be associated with further lesion of the cord, or with adventitious growths of the vertebral column or arachnoid. The only growths of this kind that are of frequent occurrence, are formations of bone on the visceral side of the latter membrane. In this respect, we perceive a characteristic distinction between the head and the spinal column; for while, in the former, ossific deposits are common in the dura mater, and are scarcely ever met with in the other membranes, in the spinal column they are found to prevail in the arachnoid, and not to affect the dura mater. A remarkable instance is recorded by Herbert Mayo, in his *Outlines of Human Pathology*, of osseous concretions surrounding the posterior roots of the nerves, and proceeding to the lower extremity. This, undoubtedly, belonged to the class of pathological products under consideration; and the case has a special interest, from its bearing upon an important law of nervous conduction. The pains suffered by the patient were so limited to the lower extremity, and were so excruciating, that the surgeon performed amputation of the limb, though with what results need scarcely be stated.

Cartilage also forms, though less frequently, on the arachnoid. A good instance is recorded in the Reports of the Pathological Society of London, by Dr. Quain,[1] who describes the laminæ as composed of a transparent matrix, in which were deposited small cells, containing nucleoli, and numerous small amorphous granules.

It does not appear that there is a liability on the part of the spinal pia mater to tubercular deposit, as we find to prevail in the cerebrum. Rokitansky remarks on the subject, that he has never had occasion to suspect the exudation formed on the pia mater to be of a tuberculous nature, and that this observation accords with the fact that spontaneous spinal meningitis so commonly coexists with that form of cerebral meningitis which produces similar exudations. An acute tuberculosis, he adds, he has never observed in it.

[1] Reports, &c. 1849, p. 25.

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CHAPTER XIII.

THE SPINAL CORD.

THE forms of disease and their effects, which present themselves in the spinal cord, closely resemble those we meet with in the brain. The spinal cord does not appear to be so often attacked as the encephalon, and as we have already pointed out, it is the part which is generally left unexamined, unless attention is forcibly directed to it by the previous symptoms of the patient; for both reasons the records of its pathological states are much more scanty than those regarding the brain, and future inquirers have yet a large field to explore. The evanescent character of congestion rarely allows of its being demonstrated after death; though it is impossible to believe that there should not be accumulation of blood in the cord, in those instances in which the symptoms demonstrate intense irritation of the part, as in tetanus; a case of hydrophobia is recorded by Dr. Bright, in which a blush of redness was perceived in the cineritious part of the spinal cord opposite the second and third cervical vertebræ. An anæmic condition of the cord is as difficult to demonstrate at its converse, though here too the practitioner will not fail to suggest instances in which its existence may be fairly assumed during life. Both states manifest themselves in the secondary effects of hypertrophy and atrophy. These may be general or local; the former affection belonging chiefly to early life, and the latter, like the corresponding condition in the brain, to old age. Atrophy of the spinal cord is one of the pathological conditions met with in the disease known as tabes dorsalis; in this case it is more of a local character, involving only the lumbar segment of the cord and the nerves passing off from that part. The separate divisions of the cauda equina are often found entirely deprived of their medulla, and nothing but the neurilemma may remain. A local atrophy, as the effect of the compression exerted by morbid growths or displacement of the vertebræ is frequently met with, and in these cases it is of peculiar physiological interest to observe the limitation of the symptoms of nervous affection according to the extent to which the pathological condition involves the

Fig. 129.

Part of a spinal cord from a case of paraplegia, with angular curvature of the spine, in a lad aged eighteen. Opposite the contracted part of the cord, a short process of bone projected from the angle of the curvature into the spinal canal.—From St. Bartholomew's Museum, Series vii. No. 7.

medulla. Numerous instances are recorded by writers on the subject; among whom we would particularly refer to Ollivier, Longet, and Romberg. Both in atrophy and hypertrophy of the spinal cord, the tissue is commonly indurated and firmer than in the normal state.

Cases of the termination of myelitis, or inflammation of the cord, in the first stage, like those of encephalitis, are scarcely ever met with; it becomes the question whether, owing to the peculiarity of the nervous structures, the first onset of inflammatory action is not at once accompanied by those changes, which in other tissues are looked upon as the secondary products of inflammation. An essential difference appears to prevail between the brain and spinal cord with regard to one of the results of congestion, hemorrhagic effusion. The frequent occurrence of apoplexy of the encephalon is familiar to all; its idiopathic occurrence in the spinal cord is extremely rare, and when brought on by external lesion, such as fracture of the vertebræ, or penetrating wounds, it is commonly associated with hemorrhage on the surface. The cases collected by Dr. Abercrombie all appear to be instances of effusion between the meninges and the cord itself. The rarity of the occurrence may justify our extracting the following observation from the Report of the Pathological Society for 1849, p. 28:—

"A gentleman, aged 44, who, with the exception of occasional attacks of gout, had previously enjoyed good health, was suddenly seized one evening with violent spasm in the stomach, and found that he had lost all sensation and power of motion in the lower half of the body. Mr. Curling found him an hour later with complete paraplegia below the third ribs, and strong priapism; no excito-motory movements were producible, and the mind was perfectly clear. The priapism subsided in about twenty-four hours; there was no extension of paralysis, except a feeling of numbness of the hands, and at last imperfect power of using them. During the first eighteen hours after the attack, scarcely any urine was secreted, and it subsequently became scanty in amount. The patient died four days after the seizure. The spine was examined seventeen hours after death. The muscles of the back were much loaded with blood—no fluid escaped on opening the theca vertebralis, the head being in a depending position. The vessels on the surface of the cord were a good deal congested. An incision was made above the front of the medulla, commencing at the part corresponding to the third cervical vertebræ, and terminating at the last dorsal; two small clots of blood, amounting together to about a drachm, were found in the interior of the medulla, occupying about an inch and a half in extent, and situated between the origins of the second and third pairs of dorsal nerves. The substance of the cord around the clots was somewhat soft; the medulla was more or less infiltrated and stained with blood from the site of the clots upwards as high as the third cervical vertebræ, and downwards as low as the last dorsal."

No microscopic examination of the parts appears to have been made; future observation must determine whether atheromatous, or other degeneration of the arteries, or previous derangement in the nutrition of the adjoining tissues, is the causa proxima of spinal, as it so frequently is of cerebral hemorrhage. The above case does not tally with the statement

of Rokitansky, that, when hemorrhage occurs in the spinal cord, it is in the cervical portion.

The product of inflammatory action most commonly discovered in the spinal cord is ramollissement, a condition which, however, like its analogue in the brain, is equally attributable to other pathological states, each of which may be recognized by the naked eye, and the aid of the microscope. The degree of softening varies from a slight diminution of consistency, as compared with surrounding parts, to a state of pulpy diffluence; the extent of cord affected differs equally. In paralysis, we very frequently meet with no other trace of disease but a trifling softening in the lumbar, dorsal, or cervical regions, manifestly the result of a slow inflammatory process. The microscopic products of inflammation, the inflammation corpuscles and granules, spoken of in considering the similar conditions of the brain, are also found in the present instance; and where any doubt prevails as to the nature of the softening, these microscopic appearances will aid our judgment. MM. Rilliet and Barthez have invariably found that, in children, the softening of the white matter of the cord coexisted with inflammation of the membranes, and that the extent of the former was in the exact ratio of the amount of the latter.

The softening affects the gray matter, and especially that belonging to the lumbar and brachial swellings more than any other part; and a case is given by Ollivier, in which the entire gray substance of the cord was converted into a pulpy mass, leaving the white matter in a comparatively healthy state. We meet with diffused suppuration in the cord as in the brain; circumscribed abscess is also, though very rarely, found within the medullary matter. Dr. Abercrombie[1] gives a case of this which occurred in a woman aged fifty-six, who was affected with sudden loss of power of the limbs of the left side, followed by death in a week. The brain was sound, but in the centre of the right column of the spinal cord, in the middle of the cervical portion, there was a cavity three inches long, and two or three lines in diameter; it was full of a soft matter, like pus, which became more consistent towards the parietes of the cavity.

It appears that the softening invariably proceeds from the gray to the white matter in myelitis. It is probable that where the process is a result of exhaustion, mal-nutrition, or degeneration, the reverse will be found to obtain; as the former would be favored by the presence of a large number of bloodvessels, the latter would spread more in a part not copiously supplied with them. The tint of the gray matter is deepened, and a rose-blush pervades the white matter in the red form, while this hue is replaced by a more or less yellow tinge when the suppurative stage has set in. A form of white softening occurs in the spinal cord which is analogous to the white softening met with in the brain, as a result of the effusion of serum or œdema, which is in no way connected with inflammation.

Another product of myelitis is induration of the cord; this is found coexistent with ramollissement, or by itself. It is more frequently

[1] Diseases of the Brain, &c., 1845, p. 355.

brought on by chronic or cachectic inflammation than softening, and it is not unfrequently complicated with hypertrophy of the affected part. When the induration is very considerable, the nerve-tissue resembles, as Ollivier observes, in consistency, density, and appearance, boiled white of egg; it is a condition that Esquirol has repeatedly met with in epileptic subjects. Gluge,[1] in adverting to the extreme difficulty of a minute analysis of all the elements of disease occurring in nerve-matter, alludes to the coagulability of the contents of the tubules as a point of great importance, though he admits the obstacles that oppose themselves to a determination of such relations in disease. He is inclined to think that the coagulation takes place in certain diseases, such as tetanus, during life. That a change in the contents of the nerve-tubules must materially affect their conducting power, is too manifest to require enlarging upon theoretically; still, the proof has, as yet, evaded our means of demonstration.

Softening of the spinal cord occurs, as in the brain, as a sequel of morbid growths, such as carcinoma, or tubercle. Neither of them is, however, frequently met with, though they are oftener seen in the cord itself than its membranes. Ollivier, whose work contains the largest collection of cases of this kind on record, denies the occurrence of melanosis affecting the cord, nor have we been able to discover any other instance but the one already alluded to, in which the melanotic tumor was attached to the dura mater.

Acephalocysts are also met with in the spinal cord. Rokitansky states that he has repeatedly met with the cysticercus in the cervical portion of the spinal marrow; but his experience agrees with that of Ollivier, that they do not occur in the substance of the medulla. They in most instances are situated externally to the dura mater. In this case, it is manifest that they had been first developed outside the column, and had forced their way in through the intervertebral foramina; they have, however, also been found within and underneath the arachnoid. It is a curious fact, for which we can offer no explanation, that the cases of acephalocyst occurring in the spinal column, have all been females.

In concluding the subject of the pathological anatomy of the spinal cord and its membranes, we must again express our regret that our knowledge of the morbid changes occurring in it are in no way commensurate with the importance and dignity of the organ. With reference to no other part of the body are we so often at a loss to explain the connection which exists between the symptoms of disease, and the actual pathological condition of the organ. The hyperæsthesia of the spinal cord (which is manifested in so marked a manner in tetanus, hydrophobia, hysteria, and poisoning by strychnine) is a palpable derangement of its functions; and yet the anatomist can discover no satisfactory reason to satisfy his desire for establishing the etiological relation. A comparison suggests itself between these cases of diseased action which appear as yet to be out of the reach of science, and those calamitous accidents on our railways which generally seem to be due rather to some

[1] Atlas der Pathologischen Anatomie, Lief. xx. p. 12.

Deus ex machinâ, than to those physical laws which are generally supposed to rule mechanical appliances. Had we desired to enter into theoretical discussion, either regarding the brain or the spinal cord, much more might have been said on the subject; and we have carefully abstained entering into the debated ground of hypothesis, as, for instance, with regard to the participation of these organs in fever, as we should thus have outstepped the limits which the practical character of this work seems to impose upon us.

CHAPTER XIV.

THE NERVES.

It very rarely happens that individuals die of an affection residing solely in the nerves; consequently, we are left to surmise their morbid appearances in those diseases in which they are manifestly affected, from analogy. At the same time, we must never forget that the nerves are not central organs, but that they are the telegraphic wires destined to convey intelligence to and from the central organs. When, therefore, we have to deal with a nervous symptom, we must first inquire whether it be due to a centric or to a peripheral cause; or, in other words, whether the nerve is propagating a morbid impression from the brain, the spinal marrow, or the sympathetic ganglia which may simulate peripheral disease, or whether it is giving evidence of local disease by producing in the brain the consciousness of that affection. In the majority of instances of irregular or painful action of the nerve, we should be as much in error in seeking for the cause of the derangement in the nerve itself, as if, when our galvanic battery does not act, owing to the trough containing no acid, we sought to remedy the defect by changing the conducting wires.

The nerves have repeatedly been made the subject of inquiry in diseases, in which either local symptoms predominate, or in which, from the known physiological action of the nerves, controlling the parts affected, the morbid phenomena could fairly be sought in an individual nerve; thus, the sciatic has been subjected to examination in individuals who had been affected with sciatica, and the vagi have been explored as the hypothetical excitants of hooping-cough. Pathologists have, in neither case, succeeded in demonstrating a relation between the malady and an uniform alteration in the respective nerves. A case has long been transcribed, and has thus acquired traditional importance, by which Cotugno, the first who wrote on sciatica, is made to affirm a lesion, œdema of the nerve, as the causa proxima of that malady; but although he records a case of the kind, he himself would certainly not have approved of the interpretation which has been given to it, since he distinctly states that he attributes no importance whatever to the circumstance. With regard to hooping-cough, we find instances recorded of the vagus having been reddened and swollen, indicating inflammatory action, but the large majority of cases in which the point has been attended to have presented no such change. Thus, Dr. Albers examined the vagi, in forty-seven children who had died of hooping-cough, and found them perfectly normal in forty-three; Dr. West, who has also

paid especial attention to the subject, has, only in one case out of eighteen, met with any change in the nerves; in this case they were decidedly redder than usual. We are inclined to conclude, with the latter author, that an appearance so frequently absent cannot be one of much moment, and that, like Cotugno's famous case, to which we have just referred, it may be set down to a cadaveric change. In our examinations of nerves, supposed to be diseased, we must be careful to distinguish between the neurilemma and its alteration, and the nerve-tubes; thus, in the stump of an amputated limb, we commonly find the nerve terminating in a button; this is owing to the effusion of plastic matter, which serves as a protection to the divided nerve, and not to an hypertrophy of its tubules.

Fig. 130.

Portion of a cerebrum with the optic nerves and remains of the left eye. The cornea is opaque, and the coats of the eye are collapsed. The left optic nerve is considerably diminished in size between the diseased eye and the optic commissure. Behind the commissure, the nerve on the right side is rather smaller than that on the left, but the thalami appear to be of equal size. From St. Bartholomew's Museum, Series viii. No. 5.

The morbid condition most commonly seen in the nerves throughout the body is atrophy; this, however, can rarely be said to be a primary affection; it is brought on by the influence of pressure, acting immediately upon the nerve, and causing gradual, and even entire absorption at the point upon which the pressure acts; this we find occurring in the case of aneurism, or enlarged glands, lying in the vicinity of nerves. Atrophy of the nerve results from the part to which it is supplied ceasing to perform the functions for which it receives the nerve; thus, atrophy of the optic nerve may follow destruction of the eye, by mechanical injury; or the nerve of an extremity wastes, when the muscles of the part are condemned to inactivity. A case is related by Swan,[1] in which, however, other nerves, the vagi, appear to have been idiopathically affected with atrophy. An individual had, for eighteen months, been unable to satisfy his appetite; the food was vomited four hours after being taken, without showing any signs of digestion; respiration became laborious and sibilant; emaciation and death ensued. At the section, the lungs were found normal, but the vagi, from the middle of the neck, were atrophied, and their terminations in the œsophagus red and thickened. The left was found smaller than the right. Mr. Swan adds that, in two consumptive patients, he found the vagi smaller than usual. In cases of atrophy and degeneration of the spinal cord, the nerves passing from the diseased portion are, necessarily, in an atrophic condition: thus, in an instance given by Cruveilhier,[2] in which the disorganization of the cord was limited to the posterior strands, extending from the lower end to the cerebellum, the posterior nerves were entirely atrophied and converted into transparent threads, which contrasted

[1] Treatise on Injuries and Diseases of the Nerves. London, 1834, p. 174.
[2] Anat. Pathol. Livr., xxxii. p. 19.

strongly with the normal appearance of the anterior nerves. It would appear that an idiopathic atrophy is met with in the nerves of sense, at least the acoustic and optic nerves have been found atrophied, in cases of blindness and deafness, where none of the ulterior causes alluded to were traceable. Rokitansky states that, under certain circumstances, nerves which are extremely atrophied acquire a grayish, translucent appearance, especially within the skull, and that the coloring is produced by the presence of a blastema filled with numerous nuclei, which, at first gelatinous, and afterwards tough and elastic, takes the place of the nerve-tubes as they disappear; it becomes more distinctly visible, as the original neurilemma of the affected nerve diminishes. He adds, that the vessels of a nerve in this condition are often palpably dilated.

Whether true hypertrophy of the nerve ever occurs is a matter that scarcely bears direct proof; *à priori*, we may assume that a nerve enlarges in proportion to the functional activity of the organ to which it belongs; an hypertrophied muscle is only enabled to manifest its power if its nerve be also hypertrophied. In such a case, there is probably an increase in the nerve-tissue itself. Enlargements of a different kind are found in nerves which are traceable to the neurilemma, or to a fibrinous deposit within the latter, as in the case of tumors of the nerves, following injury or division. After our preliminary remarks on the pathology of the nerves, we need say little about the appearance presented in congestive or inflammatory states; it is questionable whether they are ever subject to any idiopathic affection of the kind, and when the surrounding parts are involved, these invariably attract much more attention than the nerve itself. Moreover, the statements on record, though but scanty, do not agree; thus, to return again to the vagus, which has always been the pet nerve of pathologists, probably owing to its size and superficial site, we find that Kilian has observed it to be inflamed fifteen times, in pertussis, while Breschet has only met with the occurrence twice, a relation that is the more surprising when we recollect that the sphere of observation of the former is a small German provincial town, and that of the latter, the capital of France. Autenrieth, also, states generally that he has found the vagi inflamed, in persons who have died of spasmodic cough. The neurilemmatous sheath is the part mainly affected in inflammation; it presents an increase of redness, of more or less intensity; the infusion of serum induces a fulness and swelling of the nerve, and the nerve-tubules themselves become separated, and, as it were, unravelled. The exudation of fibro-plastic matter follows, and, by compression of the fasciculi, may cause their obliteration; or, if resolution ensues, the nerve may be restored to its primitive condition, or again the part may accommodate itself to the change, and the nerve remain permanently enlarged and somewhat nodose. After partial or total division of a nerve, these changes are liable to occur, and in an irritable constitution the deposit of lymph continues to act as a source of irritation, and induces intense pain. The occurrence of suppuration within the sheath necessarily gives the nerve a yellow color, and causes the tubules to be broken up. In all inflammatory affections of the nerves, the cellular tissue surrounding them will likewise be found inflamed. Two cases of inflammation of the nerve

are given by Mr. Curling,[1] in his treatise on tetanus, in which healthy spots were found, between which the nerve-tissue appeared inflamed. M. le Pelletier[2] has also published several cases, in which the inflammation appeared propagated along the injured nerve, to the spinal cord, in the same disease. The most complete investigation, however, has been made by Froriep;[3] in seven cases of tetanus, in which injury of a nerve had preceded, he has discovered a uniform lesion, resembling that indicated by Mr. Curling, and consisting in a tumefaction and reddening of isolated tracts, extending from the wound to the spinal cord; he has not found it in other cases, in which no tetanic symptoms prevailed. We have already had occasion to observe that there is no uniformity in the post-mortem appearances in the central organs of the nervous system in tetanus; it is satisfactory, at least, to know of one symptom which appears to be established, and which, at the same time, may afford an indication with regard to the necessity of local treatment, in preventing the propagation of the irritation from the peripheral parts to the central organs.

NEUROMA.

Of the morbid growths found in nerves, the so-called neuroma is the most common. In the idiopathic form, it is a growth which occurs

Fig. 131.

A stump of the upper extremity, showing the bulbous termination of the median, internal cutaneous, musculo-spiral, and musculo-cutaneous nerves; the circumflex passes behind the teres major and its termination is not seen. —From St. George's Museum, A. 35.

Fig. 132.

Neuromata of stump, after amputation of the arm. A large neuromatous mass at *a*; opposite *b*, the tumors are more defined.

within the sheath of the nerve, but does not in any way fuse with the nerve-tubules; it forms on the neurilemma, and, by gradual expansion,

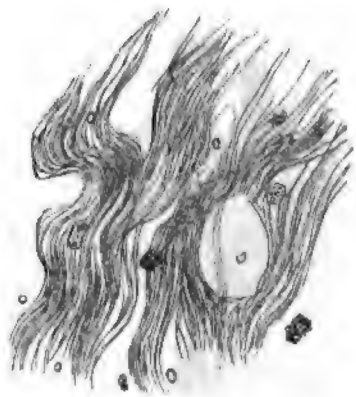
[1] A Treatise on Tetanus, 1836, p. 72.
[2] Revue Médicale, 1827, vol. iv. p. 183.
[3] Neue Notizen aus dem Gebiete der Natur und Heilkunde, 1837, vol. i. No. 1.

separates the nerve-fibrils from one another, which, with care, may always be traced from the upper to the distal part of the trunk of the nerve. We have already alluded to one kind of neuromatous formation—the button found at the termination of the divided nerve in

Fig. 183.

Section of a neuroma; three nervous trunks terminating in it. The fibrous arrangement shown, as observed by the naked eye.—Smith.

Fig. 184.



Fibrous structure of neuroma; from the case published by Dr. Smith, of Dublin. After immersion in spirit which has caused corrugation of the granules and corpuscles.—From a drawing by Dr. Bennett.

stumps. In this case, the nerve necessarily ends in the tumor, the fibrous tissue of which the swelling is composed blending with nerve-tissue, which it is destined to protect from external injury. If this view is correct, and we are supported in it by Mr. Langstaff, the swelling can scarcely be looked upon as morbid, but rather as the evidence of the curative efforts of nature. The idiopathic neuroma occurs without any known cause, in the shape of a round or oval tumor, varying in size from a grain of wheat to that of a pumpkin, and in number from one to several hundred. It must be classed among the non-inflammatory growths. The tumor is generally solid throughout, though occasionally it contains a cavity. It has a tendency to increase in size, and the nerve-fibres are proportionately distended and separated. They may generally be easily detached from the nerve. On section, the texture is found to be dense and homogeneous, closely resembling that of a fibrous tumor in other parts of the body; presenting under the microscope a fibro-cellular structure, the fibres being arranged in bands or loops, in which oval or elongated nuclei become apparent on the addition of acetic acid. Sometimes the tumor is of an atheromatous character. Dr. Smith[1] has published two cases of neuroma which are instances of the occasional extravagant production of these growths; in them, almost every spinal nerve was closely studded with neuromata, which did not,

[1] A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma. By A. W. Smith, M.D., &c., Dublin, 1849.

however, give rise to much uneasiness; in fact, it appears, as also noticed by the same author, that they are rarely productive of much pain when in great numbers; whereas the solitary neuroma, which is

Fig. 135.

Fig. 136.

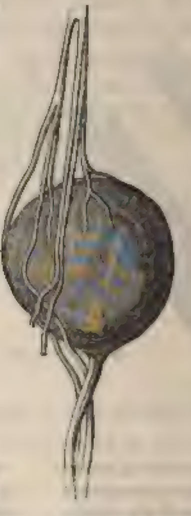


Fig. 137.

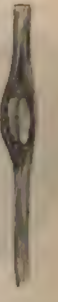


Fig. 135.—A posterior tibial nerve, in which there is a circumscribed oval tumor, composed of a soft grumous substance. The component fasciculi of the nerve are separated and spread out around the tumor, the peroneal nerve is adherent to the surface of the neurilemma, extended over the tumor.—From St. Bartholomew's Museum, Series viii. No. 1.

Fig. 137.—A median nerve, in which is imbedded a small tumor, over which the filaments are spread out. St. Bartholomew's Museum, Series viii. No. 13.

generally known by the name of painful subcutaneous tubercle, is characterized by agonizing pain. Other adventitious growths do not appear to affect the nerves of the spinal system primarily; they are involved in cancerous degeneration, by the extension of the disease from adjoining tissues; and in the nerves of the senses we also meet with the primary formation of cancer. In the retina, medullary carcinoma is not unfrequently found unassociated with cancerous growths in any other part of the system.

CHAPTER XV.

THE SYMPATHETIC SYSTEM.

A FEW cases are on record in which the ganglia of the sympathetic system were found more or less deranged and altered in structure. It is probable that a series of nervous centres, like that presented in the sympathetic, are much more frequently diseased than we have it as yet in our power to demonstrate. We can scarcely conceive that the so-called functional derangement of the heart, for instance, can continue as it does, for a series of years, and the nerves controlling its action not be or become organically altered; in the same way, long-standing derangement in the nutritive and secretive functions of the abdominal viscera may be assumed to give rise to material changes in the cœliac and semilunar ganglia, as the numerous diseases of the generative organs can scarcely exist without a similar influence being exerted upon the spermatic plexus. The anatomical disposition, as well as the physiological manifestations of the range of action of the sympathetic, justify our belief in its great and powerful agency in disease; the actual demonstration of the fact is, however, yet reserved for future inquirers. Bichat[1] repeatedly examined the nerves of the viscera in different diseases without discovering any lesions. With the exception of a single case, he has found the semilunar ganglion intact in cancers of the stomach. In a case of periodic mania, he found this ganglion of the size of a small nut, with a cartilaginous centre.

Several authors have reported cases in which one or more of the ganglia of the sympathetic were found congested and inflamed in tetanus. Swan[2] has noted this condition in each of the three cases in which he made a post-mortem examination of tetanic individuals; in the first, he found a very distinct inflammation of the semilunar ganglia; in the second, it is described as a remarkable redness, which appeared, throughout, to be produced by a very minute injection of the cellular tissue, interspersed between the small grains of which the ganglia are composed; and in the third, he states that there was an enlargement, and a greatly increased vascularity of all the ganglia of the sympathetic nerves, in the chest, and also of the semilunar ganglia; in several of those in the abdomen, the same appearance existed, only in a less degree; but in some there was neither the least redness nor enlargement.

That this, however, is not a uniform lesion, and not noted because overlooked, or not attended to, is proved by a case of traumatic tetanus, given in Dr. Bright's Reports. (Case cclxxvi.) A boy had been injured

[1] Anatomie Générale, i. 225.

[2] On Tetanus, p. 325.

in the heel by a circular saw, and had suffered a fracture of the left humerus; a week after the accident he was seized with tetanus, and died, forty hours from the first appearance of this affection. The post mortem is stated to have been conducted with the greatest care by Mr. Bransby Cooper and Mr. Key, twelve hours after death, and there were no diseased appearances discoverable; the brain was most minutely examined, as was the spine, through its whole course; the same attention was paid to the sympathetic, the nerves and the ganglia of which, throughout, appeared healthy and white. The nerves were turned down to the wound in the leg, and were also found healthy. We have given this case in detail, because it bears upon all the points already discussed, regarding the pathological anatomy of tetanus; and while it proves that none of the morbid appearances pointed out by various observers are essential, it also bears strong evidence to the non-inflammatory nature of the disorder. At the same time, it does not destroy the value of the testimony which we have found in favor of frequent physical alterations in the nervous centres, only it alters the interpretation which we might otherwise put upon them. In tetanus, the equilibrium of the forces, whose balance is necessary to the due performance of the functions of the nervous system, is destroyed, and it is quite compatible with our knowledge of pathology and of physiology, that this should occur, without any primary change in the circulating organs, while, on the other hand, we are equally justified in assuming that a state of congestion and inflammation may react upon the nervous system in such a manner as to entirely alter its normal relations. In the latter case, we should, under such circumstances, still be at liberty to view the morbid appearances, induced by the vascular system, either as the cause or the result of the changes in the balance or polarity of the nervous sphere.

It appears that the sympathetic system may also, though very rarely, be the seat of neuroma. Dr. Smith gives an instance of it occurring in the cervical ganglia, and figures it; it is, probably, the same case which is described by Cruveilhier,[1] as a case of fibrous transformation, and enormous development of the cervical glanglia, and the nerves of communication passing between them. One of the tumors was two and a half inches long by one in breadth. Both authors state that their subject was accidentally discovered in the dissecting-room in Paris, and that no history of the case was obtainable.

Dr. Smith, in alluding to this remarkable degeneration of the cervical ganglia, states that, according to Schiffner and Bischoff, this condition of the sympathetic frequently coincides with idiotcy and cretinism; it is a point which requires further confirmation.

[1] Anat. Pathol. Livr. Pl. iii.

PATHOLOGICAL ANATOMY OF THE ORGANS OF CIRCULATION.

CHAPTER XVI.

GENERAL OBSERVATIONS.

UNTIL a very recent date, the pathology of the heart was one of the most obscure departments of medical science, and the ignorance of the profession on the subject was veiled by terms which implied hypothetical views of the derangements which appeared most certainly referable to this organ. The most prominent among these were angina and hydrops pericardii; the one supposed to represent the climax of functional diseases, the other of organic changes. What the discovery of the circulation, at the beginning of the seventeenth century, by our great countryman, Harvey, contributed, to illustrate the uses of the heart and the vessels, in a physiological point of view, may be said to have been equalled in pathology by the application of the physical method of research in disease and at the dissection-table, introduced into the science of medicine during the present century. So long as we did not possess definite means of tracing morbid action in the living, it was impossible to correctly appreciate the phenomena presented in the dead subject. And thus we find that the two studies, mutually assisting and elucidating one another, have gone hand in hand. Far as a modest estimate must as yet admit pathology to be, from that to which an augmented knowledge of morbid processes may lead future inquirers, it is impossible to look back to the history of this branch of science without a feeling of congratulation; for, while every step in advance raises the intellectual standard of the profession, and diminishes the skepticism which even its most distinguished members are occasionally observed to express with regard to its actual capabilities, the mass of mankind are no less benefited both by the increased means of avoiding and preventing morbid influences, and of checking their progress when once they have fixed upon the system. But while we have succeeded in determining more uniformly and positively the locale of disease involving the central organ of the circulation, we have also discovered what may be called a type of the relation existing between the poisoned condition of the blood, to which we are justified in referring the great majority of pathological

processes, derangement of function, and disorganization of structure in a part of the economy. The doctrine of metastases, a favorite hobby of some of the older writers, has been materially modified by an extended knowledge of this relation; and at the same time an enlightened humoralism has simplified the theory of disease, and has tended to bring us nearer to the *causa proxima* of numerous processes which otherwise offer no analogy or rationale. The morbid anatomist must never forget that he has to deal, not with disease itself, but only with its products; and while it is of extreme importance that he should not isolate a single morbid phenomenon found in the corpse, but take the entire complex of derangements that present themselves, before he ventures to analyze and draw his conclusions as to their origin and connection with one another, and with the symptoms produced during life, he must also remember that the very cessation of life must alter the conditions of disease, and that there will always remain much that bears upon its intelligence, which neither scalpel, microscope, nor reagent, will be able to detect when the vital spirit has fled. A certain amount of hypothesis must, therefore, be had recourse to, to establish and satisfy the legitimate demands of science; but this theory must be based upon the entire range of our physical knowledge, and only proceed within those limits which are placed by the laws of rigid induction. It is by pursuing this system of research that the proclivity of the heart to be affected in rheumatism has been established, that we have determined fatty degeneration of the heart to be a frequent consequence of depraved nutrition, or that the close relation between renal and cardiac disease has been ascertained. The post-mortem examination alone would probably have established these facts as little as clinical observation taken by itself; but the two combined and practised by the master-minds of our century have led to results which, indeed, form an epoch in the annals of medicine. It is our duty to deal mainly with the cadaveric phenomena, but it is impossible to treat them intelligibly unless, as we have throughout sought to do, we keep in view the vital relations of the various organs in disease. After these brief preliminary remarks, we proceed, first, to consider the morbid anatomy of the fibro-serous investment of the heart, the pericardium.

CHAPTER XVII.

THE MORBID ANATOMY OF THE PERICARDIUM.

THE diseases affecting the pericardium partake of a double character, owing to the two constituents which enter into its tissue, the fibrous and the serous layer; on the one hand they are allied to the affections of fascial and tendinous formations, on the other to those of pure serous membranes. The morbid process that commences in the one may be propagated to the other, and the effects of disease occurring in one may be manifested by the products peculiar to the other. We deem it the more important to insist upon this point, as it bears strongly upon the difference between the various post-mortem appearances, and because it aids us in appreciating the pathological distinctions between pericardial and endocardial disease. These are particularly manifested in their relation to the crases; while the morbid products found in the pericardium are generally evidence of active disease, the endocardium almost serves as an index for the amount of crasis prevailing in the system; at least, no membranous structure so frequently presents alterations in those constitutions which we have elsewhere considered as characteristic of blood crases.

The frequency of morbid alterations in the pericardium increases with age, a proclivity which is not marked in the same degree with regard to the internal lining of the heart, which is much more prone in childhood to take on diseased action, than we should be inclined to assume *à priori*. Congenital affections of the pericardium are very rare, and though cases of its entire absence are recorded by observers like Baillie and Breschet, the majority of instances that have been classed under this head, have been shown to be only apparent anomalies, owing to intimate adhesion between the two surfaces of the sac giving rise to the semblance of the defect. A case of undoubted absence of the pericardium, where the heart lay in the same serous sac with the left lung, has recently been observed by Dr. Baly, in a man aged thirty-two.[1] It appears that an hypertrophic condition of the pericardium may occasionally occur so early in life as to seem congenital; we allude to the so-called milk spots, which are yet considered by various authors as results of inflammation exclusively. This is the view of Mr. Paget, while Dr. Hodgkin is inclined to look upon them as the product of attrition only, as they are almost universally found on the anterior surface of the heart, at the point most in contact with the anterior walls of the thorax. The discrepancy is probably reconciled by the observation that there are two

[1] Report of Pathol. Society, 1851, p. 60.

kinds of white spots, as was well laid down by M. Bizot;[1] the one, probably, owing to an inflammatory, the other to a non-inflammatory condition of the pericardium. They are cream-colored opacities of the visceral layer, varying in size from that of a sixpence, to that of a five-shilling piece, and more; which may at times be detached from the subjacent serous membrane, to which they are then connected by cellular adhesions. In other cases they are mere thickening of the pericardium itself, or rather of the subserous cellular tissue, and cannot in that case be detached from it. The former, to use the accurate description of M. Bizot, present at their commencement the form of small transparent elevations, aggregated together with circumscribed edges, and but slightly adherent to the serous membrane. They soon lose their transparency, becoming white and opaque, but still capable of being removed without injury to the subjacent serous membrane. They occupy every portion of the heart, but lie chiefly in the direction of the bloodvessels. The second variety have no circumscribed margin, they are peculiarly white, and their greatest thickness is in the centre from where they are bevelled off in all directions; these are essentially identical with the pericardium itself, and constitute a true hypertrophy of the membrane. It is important to arrive at a definite conclusion with regard to their origin, since their great frequency influences our views in regard to the prevalence of inflammatory affections of the membrane, and assists in determining our choice of treatment. Dr. Latham includes them in his table on the relative frequency of pericarditis in rheumatism, and thus establishes a ratio different from what we should arrive at if they are proved to be of non-inflammatory origin. We are ourselves of opinion that they are to be viewed as resulting from two causes; and that while they are in many instances the results of previous inflammation, they may also represent a simple hypertrophic condition resembling the horny thickenings of the cutis. The great frequency of the occurrence of milk spots[2] in Bright's disease of the kidney, tends to prove their connection with a blood crasis, which would favor a non-inflammatory fibrinous deposit.

PERICARDITIS.

The first effect of inflammation in the pericardium is to produce a reddening and pulpy thickening of the membrane, by the congestion of the bloodvessels and interstitial effusion of serosity. A beautiful reticulation of minute vessels is visible to the naked eye, and still better under a low power of the microscope; both on the internal and external surface of the pericardium exudation on the free surfaces then follows, which, according to the constitution of the individual, is of a more or less plastic character. In the most active forms, the effused matter is a semi-fluid, organizable material of a yellowish hue, forming a reticulated or villous appearance, which must be attributed to the movements of the heart. Laennec has very correctly compared the appearance thus pre-

[1] Mémoires de la Société d'Observation, vol. i. p. 347.

[2] See Dr. Taylor's paper on the Causes of Pericarditis, in Medico-Chir. Trans., vol. xxviii. p. 468.

sented, to that we see on quickly separating two slabs of marble, between which a layer of butter was interposed. This plastic material gradually becomes organized, and we find minute red vessels projecting

Fig. 138.

A heart covered with plastic exudation, investing both the parietal and visceral layer of the pericardium, which has been cut open and reverted. An incision has been made through the false membrane over the left ventricle, to turn it back and show the subjacent muscular tissue. The lymph fringes the right auricle and coats the root of the aorta.

into it; and as this process proceeds the two surfaces become intimately adherent to one another; the lymph loses its fluid constituents; it is converted into firm bands, connecting more or less loosely the visceral and parietal pericardium, which, according to their density and tenacity, indicate the period of their formation. If adhesion does not result, absorption may remove these appearances, and nothing but a general opacity or thickening of the pericardium remains; or again, the active condition may be arrested after the formation of villi, and without the supervention of adhesion they may continue in a passive state, and present the appearance termed the hairy heart, the cor villosum. It is customary to quote as an instance of this a classical name; the great enemy of Sparta, Aristomenes, was captured and killed on his third entry into Lacedæmon, and his heart is stated by Pausanias to have been found covered with hair. In a less sthenic constitution the effusion resulting from pericarditis will be of a more serious character; and we then find the pericardium more or less distended with a straw-colored fluid, in which flakes of lymph are discovered, while traces of lymphatic exudation are seen attached to the membrane with thin free ends waving in the fluid. We have seen the pericardium mount up from this cause to the second rib, and the quantity of serum varies from half an ounce to two quarts. In this case, as in the former, absorption may take place, leaving but comparatively slight traces of the previous disease, and the pericardium itself appears to adjust itself to the reduced quantity of its contents.

A third form of exudation met with is of a purulent character, which

is of a more atypic nature than the last. It is the least frequent, and is always associated with a large amount of serous effusion. It is chiefly met with in protracted cases, though Dr. Hope avers that even in the first stage a degree of milky opacity is observable in the serum, which may be attributed to an admixture of real pus. Hope is of opinion that even pus may, if not exceedingly copious, be sometimes partially absorbed, leaving only its solid parts to undergo ulterior changes.

The serous effusion just spoken of must not be confounded with the dropsical accumulation of fluid, to which we should restrict the term hydropericardium, and which is a frequent accompaniment of general dropsy. In many cases of wasting disease we find a few ounces of serum in the pericardium, which we must refer to mere want of tone in the vessels, and which appears to be eliminated shortly before death. It is not associated with symptoms of inflammatory action; and the fluid itself is a clear, amber-colored serum. In long-continued dropsy of the pericardium the heart is generally found contracted, and the muscular tissue anemic and of light-brown hue. Occasionally, an atrophic condition of the sarcolemma results, which is characterized under the microscope by an absence of the striation seen in healthy muscle.

In the exudation resulting from acute inflammation we occasionally meet with a small quantity of blood. Hemorrhage, independently of this cause of mechanical injury, or of rupture of the muscular tissue of the heart, is not met with in this locality as it is in the sac of the arachnoid. As regards the extent of the phlogistic process in the pericardium, it generally involves the entire surface of the membrane in acute cases; the chronic form, except as a sequel of the former, has a tendency to limitation, and its residuary effects are seen in the form of circumscribed white patches, either on the visceral or parietal portion, or of partial adhesions or isolated bands of false membrane.

Pericarditis is not often an idiopathic disease. Dr. Latham, who was the first to notice its frequent complication with the rheumatic diathesis, has rarely met with it except in this connection. Andral gives six cases of pericarditis not connected with rheumatism, of which three were uncomplicated with any other morbid affection; while Corvisart only met with five independent of rheumatism, which were all, excepting one, complicated with disease of other parts. The rheumatic complication is one found at all periods of life. Messrs. Rilliet and Barthez and Dr. West look upon it as essential in young children; and all writers on the subject concur with regard to its frequency in adults, though the statistical results arrived at are not perfectly uniform. A further powerful predisposing cause is to be found in renal disease, and more especially in that form known as Bright's disease of the kidney. Dr. Taylor gives the following results of the analysis of the causes of thirty-eight cases of acute pericarditis:—

There was rheumatism in	20 cases.
Bright's disease in	10 "
Bright's disease doubtful, or other form of renal disease, in .	5 "
Cyanosis	1 case.
Extension of inflammation from adjoining tissues in . .	1 "
	37

It follows that in examining the dead subject, we should, in all cases of pericardial disease, be particularly careful not to omit looking to the condition of the kidneys, even if the symptoms during life were not such as to draw the physician's attention to these organs. Considering the degenerative character of Bright's disease, and its chronic course, we are justified in regarding it as a powerfully predisposing cause to inflammation of serous membranes, and particularly of the pericardium. The fact of the relation of the two diseases being established, will also assist us during life in discovering one by the indications of the other, as has already been the case in regard to rheumatic pericarditis; for the subjective symptoms of the latter are occasionally so slight, that but for our knowledge of the predisposing influence of rheumatism, we might not be induced to look for the evidence of heart disease. This remark applies with almost greater force to affections of the endocardium, which, as we shall have occasion to see, offers a yet greater proclivity to the morbid influence of the rheumatic diathesis than the external investment of the heart.

The false membranes remaining after an attack of pericardial inflammation, may, unless absorbed, become the seat of similar changes, as we find them undergoing in other structures throughout the body. They present a metamorphosis into fibrous, cartilaginoid, and osseous tissue. The deposit of the latter occurs in smaller or larger patches; they may be numerous and distinct from one another, or they may unite to form, as in a preparation contained in Guy's Hospital (No. 1,448), a complete ring encircling the base of the heart.

TUBERCLE.

The relation of pericardial inflammation to definite dyscrasiæ is evinced negatively, by the absence of any proclivity of the membrane to be affected in tubercular disease. On theoretical grounds we might have been inclined to assume that the vicinity of the diseased lungs in phthisis, as well as the more immediate relation which would seem to exist between the blood circulating in the pulmonary and cardiac vessels, would have been a frequent source of disease in the latter, and the parts supplied by them. But while the meninges of the brain and the peritoneum are constantly found to be the seat of tubercular deposit, the pericardium is remarkably free from it. Louis[1] has only found evidence of pericarditis three times in phthisis; and he details one case in which some semi-transparent gray granulations were found under the serous lamina of the pericardium, to which he attributes the pericarditis under which the patient was laboring. Dr. Hope states that tubercles are sometimes developed in the false membranes of pericarditis; but neither does he himself give any positive evidence to that effect, nor have we succeeded in finding proofs of it elsewhere. It does, however, appear that the false membrane may itself become subject to simple inflammation, which, from its known vascularity, is in accordance with the general theory of inflammation.

[1] Mémoire sur la Pericardite, &c., 1826.

CARCINOMA.

Carcinoma affects the pericardium more frequently than tuberculous disease; it occurs only in connection with a general cancerous cachexia, and a formation of similar growths in other organs; the only variety met with is medullary carcinoma. According to Rokitansky, this secondary mass spreads itself in the form of an infiltration of the fibrous layer of the pericardium over a large portion of its surface, and presses upon and into the tissue itself, where it becomes developed into roundish, or flattened, or teat-like nodules.

FATTY DEPOSIT.

It is not uncommon to meet with an excessive deposit of fat upon and within the pericardium; it occurs in conjunction with general obesity, as well as in cases where there is little subcutaneous fat; nor is it necessarily associated with true fatty degeneration of the muscular tissue of the heart, though we may at the same time find fat insinuating itself into the heart, so as to separate the muscular fasciculi from one another. It will be observed that accumulation upon the heart is largest in the horizontal sulcus, and that its distribution appears to bear a relation to the arrangement of the bloodvessels.

PNEUMO-PERICARDIUM.

A condition of the heart rarely found until after death, and termed by Laennec pneumo-pericardium, consists in an effusion of air into the sac. Laennec states that he was able to diagnose its presence during life from the unusually clear sound yielded by percussion in the region of the heart, and by a sound of fluctuation accompanying the movements of the heart and of respiration. In the majority of cases, it is due to post-mortem decomposition of the pericardial fluid. The vital generation of gas in the sac must be an occurrence of extreme rarity, since Rokitansky does not appear to have met with an instance. M. Bricheteau is quoted by M. Bouillaud[1] as having met with a case of hydro-pneumo-pericardium, in which a murmur resembling the noise of a water-wheel was heard during life, evidently connected with the alternate movements of the heart. The pericardium was found to contain a fetid effusion, and, on incision, the contained gas escaped with a lisping noise. During the present year (1852), a case of perforation of the œsophagus, which had formed adhesions to the pericardium, occurred in St. Mary's Hospital. The patient was a young woman under the care of Dr. Chambers, in whom the admission of air into the pericardium occurred shortly before death through the perforation; the pericardium was found much distended from this cause, when the post-mortem examination was

[1] Traité Clinique, &c., vol. ii. p. 472.

made; and it was owing to this circumstance that the fibrinous layer, which had been deposited between the surfaces of the pericardium, had not given rise to any friction sound during life.[1]

FIBRINOUS CONCRETIONS IN THE PERICARDIUM.

To complete the subject of the morbid contents of the pericardium, we have yet to advert to the presence of free bodies, which Rokitansky has met with in a case of pericarditis. He describes them as fibrinous, soft, yellow concretions, of the size of beans or almonds, and similar to the latter in shape; which, he adds, would no doubt have eventually been converted into elastic, tough bodies of fibroid tissue. None of the authors whom we have been able to consult, record any similar case; we may therefore assume that the actual occurrence of free bodies is a circumstance of extreme rarity, and the above seems rather to be due to an accidental agglomeration of fibrinous flakes than to any other new production of tissue.

[1] The case is detailed in the Report of the Pathol. Soc. for 1852-3.

CHAPTER XVIII.

THE MORBID ANATOMY OF THE HEART.

The close relation existing in disease between certain affections of the pericardium and endocardium would be a sufficient excuse for taking the affections of the latter into consideration at once. But it appears more convenient to follow the anatomical sequence, both because there is an undoubted relation between many morbid states of the serous envelop and the muscular substance, and because the pathology of the endocardium is a natural transition to the morbid anatomy of the cardiac valves and the bloodvessels. Much has been done of late to promote our knowledge of cardiac disease, and the main result has been to withdraw many so-called functional diseases of the organ from this category, and to classify them with the known lesions met with in other tissues of the body which are more accessible to examination in life. It is more particularly in the sphere of degenerative changes, and their effects, that the advance has taken place; much yet remains to be done in the demonstration of the simple and primary forms of disease.

Congestion of the heart may be assumed to exist in the early stages of several affections with which we become acquainted, when they interfere with the vital functions. We frequently find the heart of a dark color, and the veins overcharged with blood, as secondary effects of disturbance in the circulation; in the same way, we see an anæmic condition of the organ manifested by pallor, and a flabby condition of the muscular tissue, in the train of long standing and debilitating disease.

CARDITIS.

Genuine carditis, ulceration, and abscess of the heart, are conditions of which but few cases are recorded. Of the former, Dr. Latham[1] details a remarkable instance. It occurred in a boy, aged twelve years, who presented all the symptoms of acute cerebral disease, without any indications of the disorganization found after death. No vestige of morbid action was discovered in the brain, but the heart was the seat of the most intense inflammation, pervading both the heart and muscular structure. There was the ordinary evidence of recent pericarditis, and when the heart was itself divided, the muscular fibres were dark-colored, almost to blackness, loaded with blood, soft, and loose of texture, easily

[1] Lectures on Clinical Medicine, &c., 1845, Lect. xxv.

separated, and easily torn by the fingers, and at the cut edges of both ventricles small quantities of dark-colored pus were seen among the muscular fibres. The internal lining was of a deep red color, without any effusion of lymph. A case which resembles the one just detailed, and which occurred in the practice of Mr. Salter, of Poole, is detailed in the twenty-second volume of the *Medico-Chirurgical Transactions.* It affected a man aged fifty, who died after a short illness, in which the main symptoms were oppression and distress at breathing, inability to lie down, and a dull, heavy pain at the sternum. The pulse at the same time was eighty, and regular, and the action of the heart seemed natural. There was no serous or other effusion into the pericardium, nor any signs of disease in the valves or endocardium. The heart itself was somewhat larger than natural, and its substance of moderate firmness. The great deviation from the normal condition of the heart was to be found in the muscular condition of the left ventricle. Excepting a few lines in thickness on either surface, the left ventricle had entirely lost its muscular color; it was of lightish yellow hue, but still preserving the fibrous character of muscle. From all the cut surfaces purulent matter could be scraped; in some parts absorption had taken place, leaving small cavities in the muscular substance, varying from the size of a pin's head to that of a small pea; these were all filled with pus. A third case of the same kind is related by Mr. Stanley in the seventh volume of the *Medico-Chirurgical Transactions*, p. 323, which occurred in a boy aged twelve. In purulent infection, when deposits of pus are found in the parenchyma of every organ of the body, the heart rarely presents similar appearances. It is a common thing to find the muscular tissue in severe pericarditis of an abnormally dark color, and it seems probable that, in the cases alluded to, the primary disease lay in this membrane. In those rare cases in which an ulcer is found in the substance of the heart, and an abscess occurs in the muscular tissue, we may fairly assume local inflammation to have preceded, and in the cases which are presented by the records of pathological anatomy the appearances are generally mentioned as indicating such a process. In a case of general hypertrophy of the heart, accompanied by enormous dilatation of the mitral orifice, and diseased aortic valves, which was exhibited at the Pathological Society in 1847, by Dr. J. R. Bennett, an opening of the size of a quill was found in the ventricular septum; this was surrounded by ulceration, warty roughness, and thickening, and there were distinct traces of inflammation round the opening. Hemorrhage into the substance of the heart is occasionally met with in small spots, in connection with pericarditis; but in cases of genuine cardiac apoplexy, as Cruveilhier terms effusion of blood into the substance of the heart, which must be considered in the same category as spontaneous rupture of the organ, we almost invariably find fatty degeneration at and near the point, which has destroyed the uniform consistency of the organ.

Before entering into the examination of this morbid condition, we must mention one undoubted residue of inflammation, which presents the appearance of fibrinous deposit, or an interstitial deposit of lymph. We meet with this in rheumatic subjects. The heart presents a feeble, flabby

appearance, and spots of an irregular shape and a pale yellowish hue are found scattered through the tissue. The circumference of these spots is tolerably defined, or the deposit appears to follow the direction of the fibres. It may itself be associated with fatty degeneration, but the microscope sufficiently serves to distinguish it from this condition. It is found to consist of granular matter, within which nuclei and fibroid cells are imbedded, and oil-globules. This is entirely external to the muscular fibre, which commonly exhibit an atrophic condition in the immediate vicinity of the deposit. Dr. Hope[1] recognizes three varieties of softening occurring in the heart as a result of inflammatory action: a red, whitish, and yellow form. The red, he says, corresponds to the first stage of carditis, and is analogous to the inflammatory engorgement constituting the first degree of peripneumony; the whitish corresponds with a more advanced stage, analogous to the second and third degrees of peripneumony, when a pale tint is produced by the absorption of the red particles of the blood, and by the presence of lymph and pus in variable proportions. The yellow variety he considers rather the result of chronic inflammation. These views are supported by Laennec, Bouillaud, Corvisart, and other continental authors, who at the same time admit the distinct character of a fatty degeneration. The microscope has of late contributed much to unsettling these doctrines, inasmuch as the naked-eye view has been almost superseded, and, in some instances, we fear, to the detriment of true science. For, valuable as it is in analysis, if used exclusively it prevents that general *coup d'œil* which embraces more than one morbid phenomenon, and which is necessary to the due appreciation of disease in its totality. We are induced to make this warning remark, because, in analyzing the records of the post-mortem appearances of heart-disease during the last six years, we find that almost invariably a fatty degeneration of the organ, or of individual parts, has been discovered; and this condition is one which is by no means limited to a certain well-defined disease, but is found to prevail so extensively that we cannot but look upon the molecular disintegration implied by it as a mere symptom of various elementary morbid states. It is undeniable that the appearance of minute vessels, *e.g.* of the brain in a state of fatty degeneration, as found in the vicinity of apoplectic clots, or in connection with atheroma of the larger arteries, closely resembles the appearance presented by vessels of the pia mater surrounded by or containing so-called exudation-matter in meningitis. In both instances, microscopic globules, of a highly refracting character, are the characteristic symbol. The fibre of the heart, when affected in this manner, presents a similar appearance.

FATTY DEGENERATION.

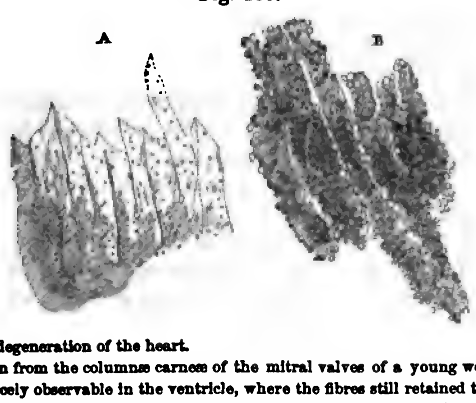
The degeneration may be traced through various stages. It commences as an atrophic condition, in which the fibre loses its sharp edges, and the striæ, so well seen in the healthy heart, disappear. One or

[1] A Treatise on Diseases of the Heart, &c., 3d edit. p. 333.

more oil-globules successively appear, until the whole fibre is occupied by them; its outline is broken, and in the highest development the fibres appear almost fused together into a confused, more or less opaque, mass, in which nothing of the original tissue can be traced. This molecular deposit of oil in the fibre is often accompanied by a generally adipose condition of the organ, and a layer of fat on the surface; but not necessarily so, and we must be careful not to infer that the heart is in a state of fatty degeneration because it is surrounded by adipose tissue. The fat may insinuate itself between the muscular fasciculi and fibres, and yet no change in the latter take place. The large circular fat-cell, with its sharp boundary of $\frac{1}{500}$th of an inch in diameter, when seen under the microscope, occupies the former position, and cannot be mistaken for the minute oil-globule, which varies from a microscopic point to $\frac{1}{5000}$th of an inch, and is confined to the interior of the sarçolemma.

The heart affected with fatty degeneration has lost the firm muscular appearance which characterizes it in health, and presents a pale, yellowish, buff color, either throughout or limited to individual parts. When cut into, a greasy stain is often left on the knife, though the absence of this circumstance must not be looked upon as an indication that the heart is not degenerated. The left ventricle and the columnæ carneæ are most liable to be thus diseased; next in order the right ventricle and right auricle, while the left auricle is least frequently involved. Dr. Quain,[1] to whom we are indebted for a clear *résumé* of the whole subject of fatty degeneration of the heart, has found that in

Fig. 189.



Specimens of fatty degeneration of the heart.

A. Heart-fibres taken from the columnæ carneæ of the mitral valves of a young woman, æt. 30; the fatty degeneration was scarcely observable in the ventricle, where the fibres still retained their striæ.

B. An extreme case of fatty degeneration, showing an entire conversion of the muscular fibre into oil-molecules, still retaining a linear arrangement. It is taken from the right ventricle of an old gentleman, who had Bright's disease of the kidney and pulmonary phthisis, and was affected with fits during the last two years of his life.

twenty-two cases, in which the seat of the disease was expressed, the two ventricles were affected in ten, the left alone in eight, and the right alone in four. Rokitansky describes three forms of fatty degeneration of the heart; the first two are varieties of fat-deposit upon and within

[1] Medico-Chir. Trans. vol. xxxiii. p. 121.

the heart, external to the muscular fibre to which we have alluded above, and to which, with Dr. Quain, we would apply the term fatty growth, while the term fatty degeneration should be confined to that change in the muscular fibre which we have just described, and which constitutes Rokitansky's third form. The deposit and the degeneration may coincide, but there is no definite relation between the occurrence of the two.

The frequency with which fatty degeneration of the heart occurs among the patients that present themselves in a London hospital, may be inferred from the circumstance that Dr. Ogle has met with it in 100 out of 143 post mortems, in which he noted the microscopic appearances of the organ; a circumstance sufficient to rivet the attention of nosologists upon the heart, in order to determine with more accuracy than we at present can bring to bear, the incipient morbid conditions to which this state is due. It is essentially a disease of middle and advanced life; and is, we may say, invariably associated with a fatty condition of other organs, more especially of the liver, the spleen, and the arterial system; in this is borne out the observation of Dr. Latham, that excepting those cases in which the damage done to the heart could be clearly traced to some distinct attack of accidental disease, his records of dissections do not supply him with a single instance of a person reputed to die of disorganized heart and its consequences, in whom, after death, other parts were not also found disorganized. And, he continues, the kind of disease in other parts has been such as could in nowise have been derived from the heart, but must have grown out of special morbid processes within themselves, whether prior or subsequent to, or simultaneous with, the disease of the heart.

Dr. Quain concludes, from the circumstances, that when muscular tissue is exposed to certain influences, such as a stream of running water or the action of dilute nitric acid, it assumes appearances identical with those of fatty degeneration, that the processes occurring in the dead meat and the living heart are identical. We are willing to admit the analogy, but we are of opinion that the uniform evidence of a degenerative tendency throughout the system, accompanying the fatty heart, is a strong proof of the ultimate cause residing in the organs and function of nutrition. The circumstance of the coronary arteries being almost invariably atheromatous, or in a state of ossification, is rather corroborative of the view, for we are not justified in assuming a primary and idiopathic affection of these vessels. Whatever may be the theory of the disorganization, its existence and frequency is sufficiently established, as well as the fact of its being the cause of further changes in the muscular tissue of the heart, which are a common source of suffering and death. Syncope and angina pectoris during life, are among the effects of fatty degeneration of the heart and diseased coronary arteries; apoplectic effusion into the substance of the organ, rupture, dilatation, and aneurism of the heart, are found in constant connection with this affection.

APOPLEXY AND RUPTURE OF THE HEART.

Without a knowledge of this change, which has taken place in the tissue, and precedes the occurrence of these accidents, it is impossible to offer any rationale for them; but now that we are acquainted with the fact that the muscular fibre is degenerated at certain parts of, or throughout the organ, it is easy to understand that, under given circumstances, requiring an unusual effort in the heart, the weakest point will yield, and give rise to effusion of blood. It is difficult to offer an explanation for those cases in which the rupture appears to have occurred during perfect rest; but it is not unreasonable to suppose that, when patients have died from this cause, while reposing in their beds, a sudden movement of the body may have been the immediate cause of the accident; much in the same way as we see, in syphilitic and other cachectic states, the bones become so friable as to be fractured from the same trifling cause. There is no essential difference between those cases in which the hemorrhage seems confined to the muscular tissue, and those in which, owing to a laceration of the pericardium and endocardium, a passage is established by which the blood flows into the serous sac. In the latter case, we find the pericardium, on opening the body, distended with fluid blood, or, if the individual has survived some time after the accident, the blood is partly coagulated. The rent varies from an inch in length to a minute orifice; it frequently runs into the septum, and occasionally we find an accompanying rupture of the columnæ carneæ. The left ventricle is by far the most frequent seat of these disruptions; we find that six of the seven cases of spontaneous rupture of the heart, detailed in the Reports of the Pathological Society of London, occurred in the left ventricle, and only one in the right. An analysis of these cases also shows that the prevailing impression that the anterior surface is more liable than the posterior to become lacerated, is erroneous; five having occurred on the posterior, and two on the anterior walls of the heart. In all there was fatty degeneration, most marked at the seat of injury; the coronary arteries were found in an atheromatous or ossified condition, in the five cases in which they were examined; the average age of the sufferers was 69¼ years. A rather different result is obtained by an analysis of cases of rupture of the heart, following mechanical injury, without penetrating wounds; here, there is no suspicion of fatty degeneration, and a different explanation must be sought for, to account for the seat of the rupture, which appears to vary as much as the injury itself. We find that of five cases of this description, in all of which there was no penetration of the heart's substance from without, one occurred on the posterior surface of the left ventricle, one on the posterior surface of the left auricle, two on the anterior surface of the left auricle, and one on the anterior surface of the right ventricle. Here, the left auricle was three times affected, and each of the ventricles once. From what has preceded, it may be gathered that we do not take Dr. Hope's view, that ulceration is the main cause of rupture of the heart; a solution of continuity of the lining membrane of the heart from this cause, is, as we

shall have occasion to mention, when considering endocardial disease, an occurrence of extreme rarity, and it does not appear to bear any direct ratio to rupture, though it may give rise to gradual perforation.

Rupture of the heart is generally immediately fatal; instances are, however, recorded, in which the patient recovered from the first shock and survived for several hours; in these cases, nature is found to have made an effort at repair, in the shape of a film of lymph, exuded between the torn surfaces.

Gangrene of the heart is a subject alluded to by pathologists, but it does not appear that any authentic cases of its occurrence are recorded. Dr. Copland looks upon it as manifestly a post-mortem alteration, accelerated by a depraved habit of body. We may, therefore, at once pass to the consideration of two conditions which are very frequent, and which are nearly allied to one another, hypertrophy and dilatation of the heart.

CHAPTER XIX.

HYPERTROPHY OF THE HEART.

In determining the existence of hypertrophy of the heart, we must attend to two preliminary points; first, we must ascertain whether there is an absolute increase of the total bulk, as compared with hearts of healthy individuals of the same age and conformation; and secondly, whether the relative size of the walls of the different cavities has altered. Next, it will be well to inquire into the relation existing between the walls of the cavity and its capacity, and it is also necessary to remove the contents, fluid or consistent, that may distend the cavities, before we form our estimate. Laennec suggested that the doubled fist of the individual might be taken as a rough measure of the size of his heart, as he found, that, in health, the two corresponded in their dimensions; there is no objection to retaining this indication, to assist our judgment, when more accurate determinations are not at our command. The weight of the healthy adult heart varies from eight to ten ounces, while, in hypertrophy, it is found to rise to as much as five pounds.

At the same time, we must also bear in mind the fact, sufficiently well determined by Bizot,[1] that there is a progressive increase in the dimensions of the heart, from infancy upwards; a circumstance that does not, at first sight, appear to tally with the general law of involution, but will, in many instances, probably, find an explanation in so-called fatty degeneration. The following table shows, at a glance, the results arrived at by that inquirer:—

Males.					Females.				
Age.	Number of subjects examined.	Length of heart in lines.	Circumference of heart in lines.	Thickness of heart in lines.	Age.	Number of subjects examined.	Length of heart in lines.	Circumference of heart in lines.	Thickness of heart in lines.
1—4	7	$22\frac{4}{5}$	27	$10\frac{4}{5}$	1—4	8	$22\frac{5}{8}$	$25\frac{7}{8}$	$10\frac{1}{8}$
5—9	3	$31\frac{1}{5}$	33	$12\frac{4}{5}$	5—9	10	$26\frac{3}{10}$	29	$11\frac{3}{10}$
10—15	3	34	37	14	10—15	5	$29\frac{3}{5}$	$31\frac{1}{5}$	$12\frac{2}{5}$
16—29	18	$42\frac{6}{18}$	$45\frac{13}{18}$	$17\frac{17}{18}$	16—29	14	$38\frac{4}{7}$	$42\frac{9}{14}$	$17\frac{4}{7}$
30—49	23	$43\frac{3}{23}$	$47\frac{13}{23}$	$17\frac{4}{23}$	30—49	27	$41\frac{5}{27}$	$44\frac{1}{27}$	$14\frac{14}{27}$
50—79	19	$45\frac{17}{19}$	$52\frac{16}{19}$	$18\frac{6}{19}$	50—89	19	$42\frac{4}{19}$	$46\frac{11}{19}$	$16\frac{5}{19}$

[1] Mémoires de la Société d'Observation, tom. i. p. 262.

According to this table, there is an uniform increase in all the dimensions of the heart, from infancy to old age, with one exception, viz: in females after the age of 30, where there is a falling off in the thickness of the organ, the other dimensions continuing to increase. The heart appears, subsequently to recover itself, and again to follow the general law, but not sufficiently to attain a thickness proportionate to that in the male heart of the same age. Mr. Bizot's measurements are taken vertically, from the apex to the base, round the base at the junction of the auricles and ventricles, and at the thickest part of the left ventricle.

The part most commonly affected with hypertrophy is the left ventricle, and even when other portions of the heart have acquired an increase of size, there is still an increase upon the relative dimensions of the walls of the left ventricle. Bouillaud[1] gives the following measurements of an adult normal heart, weighing between eight and nine ounces:—

	Inches.
Average circumference at base	8 to 9
do. longitudinal and transverse diameters	3½
	Lines.
Average thickness at base of left ventricle	6 to 7
do. do. do. right do.	2¼
do. do. do. left auricle	1½
do. do. do. right auricle	1

In health, the relative proportion of the thickness of the left and right ventricles is as 1 : 3; if we bear these facts in mind, they will assist us in determining any relative changes, and the weight of the organ will establish the fact of an absolute augmentation of bulk.

Hypertrophy occurs in three forms, to which Bertin was the first to draw attention, and his classification has been adopted by subsequent writers. In the first, which is termed simple hypertrophy, the walls of the heart are thickened, while the cavities retain their normal dimensions; the second, eccentric or aneurismal hypertrophy, presents an augmentation both of the lumen of the cavities and of the substance of their parietes; and in the third, which has received the name of concentric hypertrophy, the former is reduced, while the latter is alone increased. The last variety, probably, has no existence as a morbid condition, but is, according to the showing of Cruveilhier and Dr. Budd, a post-mortem effect, an evidence, simply, of the powerful tonic contraction of a robust heart. The former writer observed that it occurred in almost all persons decapitated by the guillotine, and the latter has pointed out that in all concentrically hypertrophied hearts the ventricle may be easily dilated by means of the fingers, and always dilates of itself, when the rigor mortis goes off. The simple and eccentric forms, then, are the two which alone constitute actual cardiac disease.

A priori, we should expect to meet with the former, very frequently, as a mere effect of stimulated nutrition, since the heart's action is so constantly abnormally increased, and its powers unduly taxed; and also because, according to Bizot, there is an uniform increase of the heart from birth to the grave; but such cases are the exception, while, in the

[1] Traité Clinique des Maladies du Cœur, vol. ii. p. 559. Paris, 1835.

majority of instances, some lesion may be discovered in the heart, or the larger bloodvessels, which, by impeding the current of the blood, gave rise to unusual efforts on the part of the heart, and thus to hypertrophy of its tissue; in the same way as we see the coats of the urinary bladder enormously augmented in bulk, when a long-standing stricture has daily called for violent contractions for the removal of the obstacle.

The left ventricle in either form of hypertrophy is the part that is most frequently affected; next in order, the right ventricle, and, lastly, *longo intervallo*, the auricles. There is not necessarily a relation between the increased thickness of the walls of a cavity and of the columnæ carneæ; the former may be themselves only thickened in some parts, while in others they retain their normal size; and again, we occasionally find the trabeculæ much enlarged, while the proper walls present but little variation. Hypertrophy of the heart necessarily alters, more or less, the relation between the thoracic viscera, a point of importance to the practitioner, as it also gives rise to modifications in the form and direction of the organ, which generally becomes more globular and spherical, while its apex is tilted up, and the long diameter occupies a more transverse direction than in health.

In uncomplicated hypertrophy, where we have to deal with no morbid product, but that of an increase in the amount of muscular fibre, the muscular tissue is of a deeper red than usual, and its consistency is increased; but the hypertrophy may be the result of a degenerative process, or a degeneration may have set up in the organ, subsequent to the hypertrophy having been established; the color may then be of a brownish tint, or present yellowish or fawn-colored spots, while the consistency is generally reduced. In the former case, we find the characters of voluntary muscular fibres more strongly marked than usual; the transverse striæ are more defined, and the edges have a sharper outline; in the latter, these characters are more or less altered, and we meet with further traces of the specific alteration. An analysis of the cases of fatty degeneration collected by Dr. Quain,[1] shows that the prevailing condition of the heart accompanying this state is one of hypertrophy, whether primary or secondary we are not prepared to determine, though it appears very probable that the change known as fatty degeneration is the result of various morbid processes, inducing a disintegration of tissue. In the thirty-three cases of Dr. Quain's first series, the heart is stated to be enlarged in twenty-one; in six the organ was of a normal size; in four the dimensions are not stated; in one there was dilatation without hypertrophy, and only in one was the heart decidedly smaller than usual.

The causes inducing hypertrophy are essentially of two kinds; "in the one kind," to use Dr. Watson's terms, "there is some mechanical obstruction to the exit of the blood from one or more of the cavities; a constricted state of the orifices is the most common condition. In the other kind, without any such mechanical drain or bar to the fluid, there is something to hinder the free and sufficient play of the organ, an ad-

[1] Medico-Chir. Trans. vol. xxxiii.

hering pericardium, it may be, or mal-position of the heart. The causes of hypertrophy may, therefore, be situated within the heart itself, or without and beyond it; but in all those cases in which the effect of the hinderance or obstacle is to detain the blood in one or more chambers, the hypertrophy will be likely to be accompanied by dilatation, and generally speaking the hypertrophy and dilatation result from disease in some part, which lies beyond the affected chamber in the order of the circulation." The largest hypertrophied heart, however, which we have met with, was one of the latter class, in which no such obstacle could be discovered. The specimen, which was taken from a middle-aged man, is preserved in the St. George's Hospital Museum, and weighed, when removed from the body, 5 lbs.; the left ventricle is enormously hypertrophied, and very much dilated at the same time; but beyond this increase in the size of the heart, no morbid appearances are perceptible or on record; the valves are all perfectly healthy.

Insufficiency of the valves, by whatever cause produced, is one of the most frequent excitements of hypertrophy; and as the former lesion prevails most on the left side, it agrees with the fact that the left ventricle is the part most commonly affected. Indirectly, the passage of the blood from the right side may be thus influenced, and an increase of the walls of that part result. Impediments occurring in the course of the arterial system at a greater or less distance from the heart act in a similar manner, but, as may be supposed, in the ratio of their proximity to the heart. It is thus that we account for the complication of hypertrophy, more especially of the left ventricle, with aneurisms. Obstructions in the capillary circulation, though less frequently, give rise to hypertrophy; this cause is found to obtain chiefly on the right side of the heart, partly, we may fairly conclude, on account of the greater vicinity of an extensive capillary system in the lungs to this portion of the organ than exists in relation to the systemic side. The most marked case of hypertrophy of the right ventricle which we ourselves have witnessed, occurred in a child of one year and a half, who had been long subject to pneumonic attacks; and in whom, after death, the whole of both lungs were found studded with small lobular abscesses. The substance of the right ventricle was increased in thickness by one-third, as compared with the left ventricle. An undeniable influence must be also attributed to inflammatory affections of the endo and exo-cardium, as well as to chronic inflammatory conditions of the muscular tissue of the heart; though the actual demonstration of the latter is a point yet to be effected, unless we assume that fatty degeneration may be the result of a phlogistic process. The manner in which pericarditis gives rise to it, is by causing partial or general adhesions, and thus preventing the free contraction of the muscular tissue. Endocarditis most commonly gives rise to hypertrophy, by inducing changes in the valvular apparatus, and thus affording impediments to the sanguineous current.

In the thirty-fifth volume of the *Medico-Chirurgical Transactions*, Dr. Barclay publishes a list of ninety-two cases of heart disease, taken from the post-mortem records of St. George's Hospital, of which we have made an analysis with regard to the relation of frequency of hyper-

trophy and dilatation; and we find that while the muscular tissue of the heart presented a state of hypertrophy only in twelve cases, there was hypertrophy combined with dilatation in forty-six, and only fourteen in which the latter condition was observed alone.

DILATATION OF THE HEART.

If in hypertrophy of the heart we in many cases see an effort of nature to adapt the organ to peculiar requirements entailed upon it by morbid conditions, and therefore would, *à priori*, expect to find it frequently accompanied by dilatation, we fail to discover this tendency where the latter condition occurs by itself. This form of dilatation is necessarily associated with an attenuated state of the muscular parietes, and may, therefore, be considered as synonymous with atrophy of the organ. Hypertrophy, with dilatation, corresponds to the condition to which the older authors applied the term active dilatation; while dilatation, associated with a diminution of the fleshy parietes, was known as passive dilatation. The two conditions have also been respectively called, by Corvisart, active and passive aneurism of the heart. These various terms show that the subject itself has not been established on a settled basis; and they certainly only tend to increase the embarrassment of the student. The less our nomenclature involves disputed theories the better, and as long as we are unable to base our terminology upon a knowledge of the proximate causes of disease, it is wiser to employ names that are derived from the most prominent symptom. In the present instance, we shall continue to use the term dilatation to designate a distinct class of morbid changes in the heart, and we shall separately consider the two varieties under which it occurs, the general and the partial form.

When, as Bouillaud remarks, the blood ceases to exert its stimulant and irritant influence upon the heart, and there are causes tending to enlarge its cavities, we find dilatation without hypertrophy; the blood in that case does not augment the molecular nutrition of the organ, but appears to act simply according to the laws of hydrostatics as a forcing power. That it should, however, at any time be able to cause a change in the cardiac cavities, necessarily presupposes an alteration in the power of the heart, and in the cohesion of the muscular tissue. The ultimate cause of these conditions may be supposed to reside in the nervous or vascular system, or in both conjointly.

The parietes of a dilated heart may be attenuated to an extreme degree; the thickest part of the left ventricle may be reduced to two lines in diameter, while at the apex the muscular substance may have disappeared entirely, so that the endocardium and pericardium are in opposition; at the same time, we find a corresponding diminution in the thickness of the columnæ carneæ. In one point there is a characteristic difference between hypertrophy and dilatation, independently of the nature of the lesion. This is in regard to the part affected. The left ventricle is most frequently attacked with hypertrophy, while we meet with dilatation most commonly in the right ventricle. It has been stated that the female sex are most prone to dilatation, and males to hypertro-

phy; we do not find this to be the case. On analyzing the ninety-two cases of heart-disease collected and reported in the *Medico-Chirurgical Transactions*, by Dr. Barclay,[1] with a view to determining this question, we find that, of fourteen cases of dilatation, six occurred in females, and eight in males; of sixteen cases of hypertrophy alone, seven were women, and nine men; of forty-six cases of hypertrophy combined with dilatation, seventeen were females, and twenty-nine males. On a rough average it therefore appears that in each variety there is a preponderance of about one-third on the side of the latter. Dilatation, as Dr. Hope remarks, takes place more in the transverse than in the longitudinal direction of the ventricles, and it accordingly communicates to the heart an unusually spherical form, the apex being rounded off in such a manner as frequently to be scarcely distinguishable.

Dilatation affects the auricles more frequently than hypertrophy; however, we must be careful in not hastily assuming a diseased condition, where its semblance is owing merely to distension. This is particularly the case with the right auricle, which very commonly appears much dilated, owing to the accumulation of blood taking place on this side of the heart *in articulo mortis*. If on removing the contents the cavity presents its normal appearance, we consider it to have been merely mechanically and temporarily distended; if the enlargement is persistent, we may look upon it as the result of morbid action during life. A dilatation of the right side of the heart is met with in connection with patency of the foramen ovale. A case of this kind was exhibited by Dr. Lloyd,[2] before the Pathological Society. It occurred in a boy aged sixteen, who had been subject to bronchitis, with temporary cyanosis. The right auricle was much dilated, and the right ventricle was dilated and hypertrophied, while the left side presented the normal appearances.

The preceding remarks apply mainly to general dilatation; partial dilatation, or aneurism of the heart, is, according to Rokitansky, whose views on this subject are particularly lucid, a condition depending especially upon an inflammatory state of the endocardium and the muscular tissue at the point affected. The following abridged view of this writer's opinions is the best account of the matter that we have to offer to our reader. Rokitansky assumes the existence of two distinct forms of cardiac aneurism. The first—the acute and rarer form—depends upon a laceration of the diseased endocardium, and adjoining muscular tissue, through which the blood passes; and the power of resistance being diminished at the point, a pouch is established, a fringed margin of endocardium is found at the entrance, and the blood deposits its fibrin within, while the margin becomes fringed with vegetations. Rokitansky has never seen a case in which the walls of an aneurism, formed in this manner, had become consolidated into a fibroid, callous tissue. In all the cases examined by him, the aneurismal formation was of recent date, having existed only a very inconsiderable period after the endocarditis, during the continuance of which it had originated. The second form

[1] Medico-Chir. Transact. vol. xxxv.
[2] Reports of Pathological Society, 1848, p. 223.

is the more remote effect of an inflammatory condition of the investing or lining membrane, or of the muscular parietes of the organ. This induces the development of a fibroid tissue, replacing, or, we should rather say, causing the absorption of, the muscular fibre. The new tissue contracts, the parietes lose their power of resisting the pressure of the blood, and a circumscribed dilatation ensues.

The aneurism varies much in size, from that of a pea to that of the heart itself; it does not necessarily present an enlargement at the surface, nor does it appear to possess an inherent tendency to increase; thus, in a case presented to the Pathological Society by Dr. Jenner,[1] in 1848, an aneurism was found of the size of a filbert at the apex of the left ventricle. Though there was no muscular substance over the extremity of the sac, it did not project beyond the external wall of the ventricle. The existence of the aneurism in this instance was traced back to two years previous to the death of the individual. A few old

Fig. 140.

Aneurism of the left ventricle, formed by dilatation of a circular portion of the anterior wall, in a girl aged 19. The pouch was filled with a laminated coagulum; its mouth was narrow, round, and smooth, and its parietes consisting apparently of endo and pericardium, with small deposits of a soft yellowish substance between them. The disease had probably commenced 18 months before death.—St. Bartholomew's Museum. Series xii. No. 53.

slight adhesions binding the apex of the ventricle loosely to the free pericardium, indicated a previous inflammatory condition at the affected point. It appears that the healthy tissue of the organ possesses in many instances an inherent power of neutralizing the evil effects which might be expected to result from a local loss of resisting power in the parietes; otherwise, it is difficult to understand how a cavity can be hollowed out in their substance without seriously impairing the contractile power. We sometimes find cases in which the thinning is not quite so definitely circumscribed as Rokitansky describes it, and where it yet proceeds to an extreme degree. Thus, we have seen a case of enormous dilatation of both ventricles without hypertrophy, in which the apex of the left ventricle was thinned to the size of a sixpence (St.

[1] Report of the Pathological Society, 1848–9, p. 89.

George's Museum, 1842–62). Partial aneurisms are not necessarily solitary, but there may be two or three, which may, as they progress, intercommunicate. Next to the apex of the left ventricle we find its base, and, third in order, the septum ventriculorum liable to be thus affected; in the latter case the bulging is towards the right side of the heart. Considering that there is a point in the upper part of the septum, at which normally there is no muscular tissue to maintain the separation of the two cavities, we should be led to expect the occurrence of aneurism more frequently at this point; but in ordinary circumstances the balance of the circulation suffices to prevent this result. When this form of aneurismal dilatation extends to such a degree that an opening and communication between the ventricles results, we have to deal with what Dr. Thurnam has termed the varicose aneurism; it is a condition analogous to a congenital state, not unfrequently met with, in which the ventricular septum is more or less defective.

The contents of the cardiac aneurisms vary; we find in them fluid blood, more or less decolorized, sanguineous coagula, or laminated fibrinous deposits, resembling those found in arterial aneurisms, and presenting similar microscopic appearances.

The male sex and mature age offer a greater proneness to aneurism of the heart than the female sex and youth; the proportion with regard to sex is about 1 to 3; Hasse states that of forty-seven cases thirty-five were men and twelve women; and of forty-two cases, ten referred to individuals under thirty, and thirty-two to older persons.

ATROPHY OF THE HEART.

In introducing the subject of dilatation, we observed that it was essentially an atrophic condition. Atrophy of the heart is also met with in the shape of a mere reduction of size, either as a result of wasting disease or as a congenital vice. Three extreme cases recorded by Burns probably belong to the latter variety; in one instance, he found the heart of an adult as small as that of a new-born infant, and in another the heart of a female, aged six-and-twenty, was no larger than of a child of six years. Bouillaud describes a case of an old woman of sixty-one minutely, who died of acute peritonitis, in whom the heart was a third smaller than in the normal condition, or about the size of the heart in a child of ten or twelve. The surface was furrowed and presented milk spots, the remains of former pericarditis; the cavity of the left ventricle was scarce large enough to contain a pigeon's egg, and its parietes were only three lines in thickness. In phthisis, there is a uniform diminution in the size of the heart; in no other maladies is this so much the case, as illustrated by the following measurements given by Bizot[1] of the heart in the adult:—

[1] Mémoires de la Société d'Observation, vol. i. p. 277.

MALES.				FEMALES.			
Age 16 to 79 years.	Length of heart lines.	Circumference of heart lines.	Thickness of heart lines.	Age 16 to 89 years.	Length of heart lines.	Circumference of heart lines.	Thickness of heart lines.
57 Phthisical.	$42\frac{7}{18}$	$47\frac{1}{24}$	$15\frac{11}{18}$	—	$39\frac{3}{5}$	$41\frac{1}{30}$	$15\frac{1}{13}$
65 Non-Phthisical.	$45\frac{1}{31}$	$50\frac{28}{31}$	$18\frac{13}{31}$	—	43	$47\frac{14}{18}$	$16\frac{9}{28}$

MORBID GROWTHS.

The adventitious and heterologous products found in the muscular tissue of the heart are almost all pathological curiosities, with the exception of the fatty metamorphosis to which we have already alluded. Acephalocysts are occasionally met with, as also the cysticercus and echinococcus. A well-marked instance of the latter was presented to the Pathological Society by Mr. Ward, in 1847, and the preparation is still preserved in the Museum of the London Hospital.[1] In this case no trace of the entozoon was discovered in other viscera.[1] A remarkable case in which a cyst containing hydatids was found in the substance of the heart, is recorded and delineated by Mr. Evans in the seventeenth volume of the *Medico-Chirurgical Transactions*. It occurred in an unmarried female, about forty years of age, who, during the winter preceding her death, had been subject to palpitation and angina pectoris. The cavity of the pericardium was found to be coated with a layer of coagulable lymph over a small extent of its front surface; the apex of the heart was lost in a considerable tumor, apparently an elongation of the heart itself, and covered on all sides by pericardium. The new growth was found to project into the cavity of the right ventricle, was smooth, globular, and about three inches in diameter. It contained numerous hydatids from the size of a pea to a pigeon's egg; their interstices being filled up by a soft, curd-like, yellow substance. The hydatids were precisely the same as those found in the liver. The trichina spiralis has not been met with in the heart. Ossific deposits are recorded, but they seem invariably to proceed from the endocardium; and we shall return to the subject in connection with the diseases of this membrane. Both Corvisart and Hope give cases of portions of the heart being converted into cartilage: we should be inclined to assume that if these cases had been subjected to microscopic examination, the tissue would have proved fibrous rather than cartilaginous. Tubercle and cancer occur in the heart, but only when the respective dyscrasiæ are very strongly marked; the centre of the vascular, in this respect, differs much from the centre of the nervous system. Of the two, cancer is the more frequent; it occurs in the medullary and melanotic varie-

[1] Report of Pathol. Soc. 1847-8, p. 225.

ties, and either by infiltration or in the form of isolated tumors. An exceptional case, as proving the occasional evolution of cancer in the heart, is recorded by Mr. Travers.[1] On the anterior apex of the heart of a gentleman, who died suddenly with symptoms of angina pectoris, and whose body he examined with Mr. Parrott, of Clapham, he found a fungoid growth, elevated, and of the diameter of a shilling; it had the true pulpy character. There was no ulceration, nor any corresponding appearance in the loose pericardium or elsewhere. The internal membrane was unchanged, except some earthy deposits about the aortic valves.

[1] On the Local Diseases termed Malignant. Med.-Chir. Tr. vol. xvii. p. 354.

CHAPTER XX.

MORBID ANATOMY OF THE ENDOCARDIUM.

It has required the multiplied observations of numerous laborers in the field of pathology to establish the true nature and importance of endocardial affections; Laennec and J. P. Frank were among the first to draw attention to inflammatory conditions of the endocardium, but we owe the more correct appreciation of the subject to Bouillaud and Dr. Hope. The pathology of the endocardium is as significant in regard to its primary, as it is with regard to its secondary lesions. The membrane is analogous to that lining the bloodvessels; it consists of a layer of epithelium, investing a fibrous tissue, between which and the muscular fibre of the heart there is a layer of elastic cellular tissue; it is in the latter that bloodvessels ramify, and through them the nutrition of the surface-laminæ becomes affected, in disease as well as by the direct influence of the blood contained in the cavities of the heart. It is necessary to bear this double relation in mind, as, without a due perception of these facts, we shall find it difficult to harmonize our general knowledge of morbid changes with the apparent exceptions that endocardial disease brings to our notice.

A careful examination of the endocardium is necessary, before we determine positively that the appearances we find are due to pathological alterations; the earlier observations lose much of their value from post-mortem staining having been mistaken for inflammatory reddening. When the change of color, which naturally is of the palest white, and translucent, is due to inflammation, the redness cannot be washed off, nor is it rapidly destroyed by maceration; the membrane is pulpy and thickened, and in an advanced state, further products of inflammation are found beneath, or upon the membrane. Where the redness is the result of mere imbibition, we find the blood in the cavity fluid, and it occurs in cachectic individuals. The later the post-mortem examination is made after death, and the warmer the weather at the time, the more likely are we to find the lining membrane of the heart and arteries stained with blood. We rarely have an opportunity of seeing endocarditis in its first stage. Rokitansky and Hope describe inflammatory redness of the endocardium as mottled. Dr. Hope states it to be less characterized by streaks, patches, isolated unstained spots, and abrupt edges, than non-inflammatory imbibition. The discoloration is more perceptible in the arteries than in the cardiac cavity, owing to the subjacent tissue serving as a better foil in the former than in the latter. On the other hand, the next change, that of opacity of the lining mem-

brane, for the same reason shows more distinctly in the heart than in the arteries, where a transverse section will be required to demonstrate the increase of thickness. This alteration in the appearance of the endocardium also changes it in other respects; it loses its glaze, and becomes dull, relaxed, milky, and velvety. The redness of imbibition is darker in proportion to the period the blood is in contact with the parietes of a cavity, and for this reason it is observed, as pointed out by Hasse,[1] in the following descending order: darkest in the right auricle; paler in the right ventricle, with the exception of the valves of the pulmonary artery, which are as deeply colored as the auricle; still paler in the left auricle; whilst the left ventricle often retains quite its natural tint, except that the aortic valves are darker. In the great vessels, the posterior surface is strikingly dark, in comparison with the anterior.

In rare cases, an adhesion has been found effected between opposite points of the parietes. When the inflammatory process extends to the valves, the consequent change in their relation to the blood-current, gives rise to those physical signs which are of so much value in the diagnosis of cardiac affection. The laxity of tissue induced by the inflammatory process offers more or less impediment to the circulating fluid, and may even be the cause of a laceration of the cardiac lining, or of the extension of the membrane to the valves and arteries.

The first effect of inflammation of the endocardium is the exudation of lymph on its free surface; where, however, it is rarely to be found, on account of the current washing it away; its absence was an argument used by Laennec, against the assumption that the endocardium was liable to inflammation. In how far the fibrinous vegetations on the valves are the product of inflammation of the part, or a deposit of lymph exuded within the heart, or again, an elimination of fibrin directly from the blood, is a matter not absolutely decided. It appears to us that there is no difficulty in reconciling the conflicting opinions, by assuming that the valves may become the seat of these deposits, in each of the modes alluded to. We know that, if a thread be passed through a healthy artery, a coagulation of fibrin attaches itself to the foreign body; and we also know that inflammation increases the tendency to a separation of fibrin from the blood. There is, therefore, no necessity for the intervention of the coats of the capillaries to produce the effect of exudation, although the term certainly implies a transmission through a membranous expansion. We are satisfied that we have seen what is called exudation matter, within the small vessels of the pia mater, in meningitis, and, though the number of our observations are not sufficient to base any positive conclusions thereon, the fact may, *quantum valet*, aid in the present inquiry. Besides, the circumstance of the great tendency to valvular growths prevailing in rheumatic constitutions, supports the view of the frequent occurrence of deposits being effected directly from the blood. Rheumatism, more almost than any inflammatory condition, presents the character of a blood disease, and, considering the preponderance of fibrin in the elements of the blood which

[1] An Anatomical Description of the Diseases of the Organs of Circulation, Syd. Soc. Ed. p. 128.

characterizes it, and the frequent complication with heart disease, it offers a further corroboration of the view just advocated. It is, in fact, the same as that expressed by Dr. Hope, when he says that the vegetations on the valves are caused by inflammation inducing either an effusion of coagulable lymph, or by its imparting to the blood in contact with the inflamed part, a morbid tendency to coagulate.

If the discovery of lymph on the inner surface of the heart is an unusual circumstance, it follows *à fortiori*, that we still less frequently meet with a purulent effusion resulting from endocarditis. Rokitansky remarks on the subject, that although the recognition of the seat and position of pure pus, as a free product, is, in most cases, impracticable, it is not difficult to prove the extreme probability of the existence of such a process. The loosening of the tissue, the want of polish, and the felt-like character of the endocardium, are very strongly marked in the centre of inflammation, and hence lacerations frequently occur. In these cases, a purulent product mixed with the blood is generally found infiltrated into the tissue, if not at the surface of the endocardium; whilst abscesses are occasionally found to have spread themselves over a various extent of surface below the endocardium, in the cellular and adjoining muscular strata, deep in the tendons, and in the tissue of the valves.

One of the effects of endocarditis is ulceration; it is met with on the parietes of the heart, but more frequently on the flaps of the valves, where it gives rise to perforation and rupture. Perforation of the septum ventriculorum occasionally results from this cause. A free communication may thus be established, or the muscular wall may be completely destroyed; but an exudation of fibrin having taken place on the distal side, the intermingling of the contents of the two cavities may be prevented. We find the two lesions associated, as for instance in Bouillaud's[1] sixty-sixth case; the subject, a man aged thirty-seven, was seized with rheumatism, upon which pericarditis and endocarditis supervened, causing death on the thirty-first day of his illness. At the cadaveric inspection, a cauliflower excrescence of fibrin was found upon the ventricular surface of one of the aortic semilunar valves; upon which, besides a fringe of lymph along its free margin, two perforations were discovered, one at the middle of the flap, the other lower down, and communicating with a cavity in the muscular tissue large enough to contain a bean; to the right of this flap there was a red spot of three lines in diameter, in the centre of which was a small circular ulcer, sufficiently large to admit the head of a pin. Bouillaud also gives two instances in which endocarditis was followed by gangrene, a conclusion, however, which some have cavilled at. The following is an abstract of the most satisfactory of the two: The patient, a robust individual, aged fifty-six, after exposure to a draught while much heated, was seized with endocarditis, and death ensued in six weeks. The post-mortem was made thirteen hours after death, and two of the aortic valves were found indurated at their base, and adherent to one another so as to interfere with the current. The endocardium covering their base was red, thickened, and presenting an abrupt red fringe, at a short distance from the free margin. The remainder of the

[1] Traité Clinique des Maladies du Cœur, vol. ii. p. 29.

valve was ulcerated, soft, friable, of a dirty gray, and eroded; one presented a perforation. The two flaps, says M. Bouillaud,[1] closely resembled the appearance of gangrene of the cutaneous surface, with a red line of demarcation. Dr. Copland, as we have already seen, is of opinion that gangrene will only supervene when internal carditis attacks a cachectic habit of body, or when there is a septic tendency induced in the system, by a depraved state of the circulating fluids, or by impaired vital power.

A frequent concomitant of endocarditis, appears to be, according to the statistics of Bouillaud, who has met with a larger number of fatal cases of endocarditis than any English physician, the coagulation, during life, of the blood, and the organization, in the clot, of new blood-vessels; the coagulum is found adherent to the parietes of the cavity, and requires some force for its removal. It is colorless, elastic, and glutinous, and closely resembles the buffy coat of inflammation, or false membranes themselves. The symptom by which Bouillaud recognizes this occurence before death, is, a want of accordance between the pulse and the heart in point of force; the heart presenting evidence of violent excitement and action, while the pulse is small and evanescent. Gluge[2] describes organized fibrinous coagula under the name of hæmatoma, and gives an interesting instance, with the minute anatomy of the clot, which occurred in a female, aged fifty-two. The left auricle was filled with a red tumor, surrounded by a delicate membrane, in the interior of which he distinctly traced capillary vessels, forming a retiform plexus. Similar instances may be also found in the records of the Pathological Society, and in Dr. Hodgkin's *Catalogue of Guy's Hospital Museum*. The cases in which organized clots or fibrinous coagula have been found by English observers, were generally connected with a cachectic condition, analogous to what Rokitansky terms the fibrinous crasis. The surface is found more or less intimately connected with the endocardium, while the interior of the clot may, in its turn, be undergoing further changes of an inflammatory or degenerative character. The fibrin is seen to be breaking up into a granular condition; exudation or inflammation corpuscles and fibro-plastic cells may be exhibited by the microscope. This does not necessarily apply to the pus that is occasionally found within the coagulum, which is to be regarded rather as the cause than the consequence of the coagulation. The pus may be derived from various sources; Cruveilhier[3] observes that it may be generated at a distance, and be carried to the heart by the blood-current; that it may be the result of inflammation occurring in the coagulum, or that it may be the product of endocarditis; in which case it is absorbed into the coagulum, by capillary attraction. Tuberculous concretions have also been found in the clot; however they gain the position, it must be before death; changes affording sufficient proof of the independent vitality of the concretion. The older pathologists attributed a much greater importance to fibrinous coagula, or, as they termed them, polypi, in the

[1] Traité Clinique des Maladies du Cœur, vol. ii. p. 87, Observ. 87e.
[2] Atlas der Pathologischen Anatomie, Lieferung 11.
[3] Anat. Pathol. livr. 25.

heart, than they now obtain, owing to their being regarded as the immediate cause of death. It is only in rare cases that we shall be justified in looking upon them as products formed during life; in the majority of instances they are merely the first evidence of the arrest of vitality, and the incipient influences of the metamorphoses of decay. When formed during the agony, or after death, there is no adhesion to the parietes; the polypus is moulded to the cavity which contains it, and a straw-colored fibrinous layer invests a blood-clot, similiar to the buffy coat covering the coagulum of blood obtained by venesection. The view we have taken of the organized polypi is supported by Hasse,[1] who observes that the seat of morbid action giving rise to them is, in the majority of instances, remote from the heart. Under certain circumstances the blood retained after the systole in the ventricles, and impelled into the network of the columnæ carneæ, acquires the opportunity to coagulate; and one fixed point being given, it is easy to understand how constantly fresh deposits are made on the surface, causing a laminated appearance, and aiding in the process of organization.

A second form of fibrinous concretion is described by Rokitansky, under the name of globular vegetations, as round masses, varying from the size of a pin's head to that of a nut, attached by means of ramifying cylindrical or flat appendages or bands, which entwine themselves among the trabeculæ of the heart, and are of a more or less uniformly dirty grayish-red or white color. He states them to be hollow in the interior, and to contain, within a wall of irregular thickness, a dirty grayish-red or even chocolate-colored fluid, resembling cream or pus. One or more of these concretions very frequently burst, when the fluid may be seen effused into the cavity of the heart, and distributed over the recent coagula, which have been formed either in the death-struggle or shortly after death.

Rokitansky establishes a third concretion, under which he comprises all vegetations of the valves of the heart, presenting a shaggy appearance, resembling villi, forming shaggy pedicled excrescences, or offering a cock's-comb or mulberry-like appearance. They affect the free margins of the valves, the tendons of the papillary muscles, and also attach themselves to the endocardium. They float in the blood, and necessarily lie in the direction of the current.

It appears that we have sufficient evidence to believe that they may occasionally become detached and be carried by the force of the circulation as far as the first angle of a vessel offering an impediment, or until they reach a channel which is too small to permit of their transmission. Dr. Kirkes,[2] in an interesting paper presented to the Medico-Chirurgical Society, has carefully investigated the circumstance, and recorded several instances in illustration. The part more immediately affected depends, according to this author, in the first instance, upon the circumstance of the fibrin being detached from the right or the left side

[1] An Anatomical Description of the Diseases of the Organs of Circulation and Respiration, Syd. Soc. Ed. p. 127.

[2] Medico-Chirurgical Transactions, vol. xxxv. p. 281.

of the heart. In the former case, the pulmonary, in the latter, the systemic circulation will become affected. When the mass of fibrin is detached from the left side, the lodgement is most commonly effected in one of the middle cerebral arteries, a circumstance explicable by the anatomical relation of these vessels. The arteries of the spleen and kidneys appear to be liable, next in order, to similar deposits, on account of their receiving their arterial supply by large vessels directly from the heart. That the plugging up of an artery must induce a change of nutrition in the part to which it leads, scarcely requires to be dwelt upon; while it causes coagulation of the blood behind, it acts as a foreign body, exciting inflammation and exudation, or degenerative processes, as softening and gangrene. In how far such an occurrence is remediable is very doubtful, though Dr. Kirkes suggests that a breaking up and absorption may take place, or that, by a dilatation of the blood-vessel, the current may be enabled to pass it; in the latter case, we should imagine it more probable that the plug would be propelled, especially as one characteristic of this variety of deposit is that it enters into no close adhesions to the inner coat of the vessels. Twenty-one cases have been analyzed by Dr. Kirkes, in which these deposits were found, and in every one but two he found disease of the valves and of the interior of the heart. One of these was a case of cholera, in which a doubtful mass of capillary phlebitis existed in the liver; the other was a case of aneurism of the aorta, which the author looks upon rather as favoring his views. In fourteen out of the remaining nineteen, fibrinous growths were noted on the surface of the left valves, or the interior of the left cavity.

NOTE.—Since the above has been in type, we have seen that Dr. Todd is not inclined to adopt Dr. Kirkes's view, but would refer the coagulum found in the distant artery to an altered nutrition of its wall—to arteritis—and connected with a rheumatic or other morbid state of the blood. (*Clinical Lectures on Paralysis*, &c., 1854, p. 176.)

CHAPTER XXI.

DISEASES OF THE VALVES OF THE HEART.

THE estimates of different authors, with regard to the influence exerted by inflammation in producing valvular disease, have varied considerably. Bouillaud attributes nearly all changes occurring in the valves, such as altered consistency and form, fibrinous concretions, calcareous and ossific deposits, to inflammatory action, terming them the third stage of the inflammatory process; while Rokitansky, and many with him, are of opinion that they are only in part the product of endocarditis, but that the majority are the result of slow changes of nutrition, not connected with inflammatory action.

The most manifest direct results of endocarditis are white opacity and thickening of the endocardium and the lining membrane of the valves, and adhesion between the latter. Adhesions are most commonly found in the aortic valves, and this lesion must necessarily constitute a permanent and very serious obstacle to the circulation, in its turn giving rise to further disorganization and derangement, such as hypertrophy and dilatation, asthma and anasarca. In dealing with this species of malformation, it is often very difficult to determine whether it is congenital or the result of disease, especially when, subsequent to adhesion, an absorption of the partition separating the two pouches is effected, and the double valve thus converted into one. Dr. Peacock[1] has analyzed fifty cases of malformations of the pulmonary and aortic semilunar valves, among which he found forty-one of defective, and nine of excessive development. Of the former, nine were found at the pulmonic, and thirty-two in the aortic orifice. The varieties which the fusion of the valves with one another, or their adhesion to the walls of the heart, may present, are very numerous. In all cases an insufficiency of the valves must result, which both offers an obstacle to the free discharge of the blood from the heart, and fails adequately to close the orifice during the diastole, so as to prevent regurgitation. The left side of the heart generally, and especially in regard to inflammation, offers by far the greatest proclivity to disease. Very few cases are recorded in which a phlogistic process could be demonstrated on the right side. Gluge[2] gives two observations in which the tricuspid valve was thickened and rendered insufficient by this cause. We also find two instances reported in Dr. Hodgkin's *Catalogue of the Museum of Guy's Hospital* (Nos. 1401 and 1402), in which the curtains of the tricuspid were thickened.

[1] Reports of the Pathological Society, 1851–52, p. 292.
[2] Atlas der Pathologischen Anatomie, 1850, Lieferung i. Beobachtung, 12 and 12a.

In one of these there was also shortening of the tendinous cords. While the arterial valves are more subject to this species of lesion, we find the mitral valve more prone to an hypertrophy of its fibrous tissue, which is especially liable to present itself in the shape of nodulated masses,

Fig. 141.

Fibroid thickening of the mitral valve.

fringing the curtain, and in some instances closely resembling accumulations of fat. The microscope at once determines their real nature, as it exhibits, instead of fat-cells, a fibroid structure, containing nuclei and elongated nucleated cells. In connection with hypertrophy of the endocardium, we find the lining membrane of the valves also thickened; by which means it appears that, independently of inflammatory action, a secondary adhesion may be effected between the flaps. Here the aortic valves are more liable, though it is not at all unusual to find the curtains of the left auriculo-ventricular orifice opaque throughout, from the same cause.

We have already alluded to perforation of the valves, as a result of

Fig. 142.

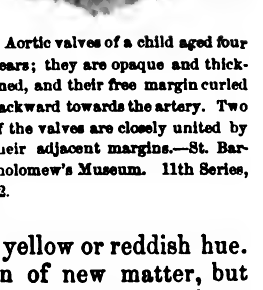
Fibroid thickening of a pulmonary valve, extending symmetrically on both sides of the curtain, and consisting of a soft fibrillating deposit. It was found in a man who had a broken spine.

endocarditis. Another form in which the same lesion occurs is in connection with atrophy. This is manifested, in the first instance, by

attenuation, and increased transparency of the valves; as this advances, one or more openings are effected, which may be sufficiently numerous to induce a cribriform appearance. It is only when the perforations are large or numerous that they interfere, to any serious extent, with the circulation. Thus, in the case of a man who died recently at St. Mary's Hospital, after an operation for popliteal aneurism, the cause of death being extensive pneumonia, there was much fibrinous deposit on the aortic valves, with two valve-like perforations, apparently the result of ulceration, which had given rise to murmurs of a peculiar character before death, but not, apparently, inducing any other symptoms of cardiac disease. Dr. Kingston,[1] who was the first to draw attention to this point, observes, that atrophy may be defined a simple shortening of the valve, and, in the first instance, a mere atrophy in the direction of the length. He speaks of the cribriform appearance in the flaps as also resulting from the same process, and has found the two conditions chiefly in the mitral and tricuspid valves. In this, he differs from other authors. Rokitansky, for instance, has only met with the lesion in the arterial valves. Dr. Kingston, out of about thirty cases of valvular disease, found the lesion to be atrophy in ten. The mitral valve was shortened in five, the tricuspid in five; both in two. In one the mitral valve was cribriform, in two the tricuspid, and in one both the aortic and pulmonary valves were so.

The lesions of the valves hitherto spoken of may be variously complicated with one another, or with heterologous growths. Pathological records contain instances of a great variety of changes of form, the result of morbid processes or accident. Thus, the individual flap of the semilunar valve may be reverted or inverted, the valves of the aorta may become detached at their bases, and thus lose the fulcrum by which they resist the impetus of the blood, or the tendinous cords of the mitral may induce a deficiency of the valve by a shortening and thickening, a lesion which Dr. Hope considers as constituting one of the worst varieties of disease of the valves.

Fig. 143.



Aortic valves of a child aged four years; they are opaque and thickened, and their free margin curled backward towards the artery. Two of the valves are closely united by their adjacent margins.—St. Bartholomew's Museum. 11th Series, 52.

Among the anomalies of consistence, Rokitansky describes, besides the increased density of thickened and shrivelled valves, and the softening that results from inflammation, a gelatinous condition of the valve which he has found in the valves of the left side of the heart exclusively. There is a loss of fibrous tissue, for which a gelatinous, non-adhesive substance is substituted, causing the valve throughout, or only in parts, to become soft and pliable while its color is converted into a pale yellow or reddish hue. The author is of opinion that there is no effusion of new matter, but that the gelatinous substance is merely the disintegrated fibrous tissue

[1] Medico-Chirurg. Trans., vol. xx. p. 90.

of the valve itself. It appears that while on the one hand this condition may lead to lacerations, especially of the valves of the aorta, pre-

Fig. 144.

Atheromatous deposit in the valves of the aorta of a man aged 26, with rupture at the point marked by *; there was also congenital union at the point (marked by †) of two of the valves. The case is described in the Reports of the Pathological Society, vol. iv. p. 100.

senting the fissured appearance of true rents, to distinguish them from the perforations resulting from atrophy, it is also susceptible of cure by a reconversion into fibrous tissue.

Fibrous and ossific deposits, which we have seen to be not uncommon on the surface of the heart, are very rarely met with under the endocardium except in connection with the valves. To the former, which are often rather hypertrophied states of the normal fibrous tissue, we must attribute many of the lesions already adverted to, consisting of malposition, eversion, and inversion, of the valves; the fibro-cartilaginous and

Fig. 145.

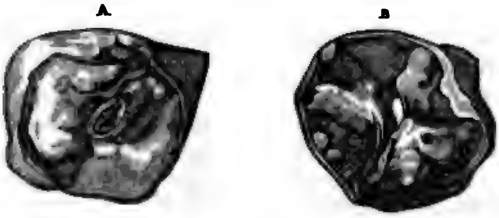
Aortic valves of a man æt. 47, rendered perfectly rigid by calcareous deposit. The patient was affected with granular kidneys and cirrhosis of the liver.

cartilaginous induration spoken of by Bouillaud and others, may be referred to this head. The calcareous or ossific deposit is a distinct new formation. It presents the most varied forms, which may be compared to the fantastic shapes assumed by molten lead when poured into water; sometimes resembling stalactitic projections, at others forming irregular rounded eminences, stretching across the orifices of the heart like rigid

bars, maintaining the valves in a state of permanent erection or distension, and inducing symptoms both of obstruction and of regurgitation. A single flap or curtain may be rendered rigid while the others retain their natural pliability: the valves of one side of the heart may be more or less intimately united by the morbid growth; but whatever forms the lesion may assume, it is scarcely possible to occur without a permanent narrowing of the orifice. Kreissig and Bouillaud refer the disease uniformly to inflammatory action; and Dr. Watson is also of opinion that it is somehow certainly connected with inflammation of the internal lining of the heart. But we must not overlook the important fact of the natural tendency existing in the arterial system generally, as well as in other tissues of the body, to induration and ossification with advancing life; and though we are far from looking upon ossification of the valves as a physiological process, we are justified by analogy, as well as by the positive fact of the very chronic nature of these deposits, in looking upon them in many cases as of a non-inflammatory character allied to the general species of degenerative disease. Lobstein's view, that these concretions are intimately allied to the gouty diathesis, is one that must not be lost sight of; though he perhaps erred in restricting them too closely to this particular constitution. Although we have used the term ossification in accordance with common usage, to designate the change under consideration, it is important not to confound the process with the one in which genuine bone is formed: cretification or calcareous deposition would be a more appropriate term, for there is no resem-

Fig. 146.

A B



Ossification of the aortic valves; a thick calcareous deposit has taken place between the valvular membranes, interposing a rigid and almost imperforate diaphragm between the cavity of the heart and the vessel.

A. Upper surface. B. Under surface.

From St. George's Hospital Museum, E 18.

blance between the morbid product and true bone. It consists essentially of carbonate and phosphate of lime deposited in irregular, amorphous nodules, and resembling more a chemical precipitation than an organic formation. The material is more or less friable, and is connected by the remains of the fibroid, or atheromatous matter, in which it formed. It is soluble in the mineral acids. It is often difficult, when we meet with an advanced case, to determine in what part the deposit first takes place; whether beneath or on the surface of the lining membrane. The opinions of different writers differ with regard to this question. The most common form undoubtedly is the conversion of atheromatous or fibroid deposit underneath the lining membrane analogous to what we

see occurring in the arteries; and as this enlarges, the membrane becomes softened and destroyed, and the ossification then projects free into the sanguineous current. It is not the mere increase of the deposit which determines this solution, but an element in producing this result is undoubtedly a morbid affection of the lining membrane itself, in which, even in early stages of degeneration of the subjacent tissue, we have observed disintegrating processes, of which we shall speak further when discussing the diseased conditions of the arterial system. One of the most extreme cases of narrowing of the aortic orifice in an adult, that we have met with, is the one delineated (Fig. 146), in which the continuity of the lining membrane was preserved entire over the ossific deposit. The passage was contracted to the size of a pea.

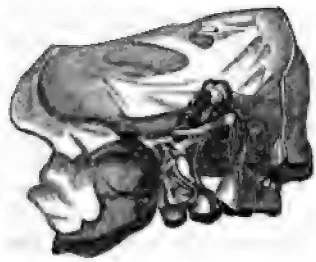
Rokitansky is of opinion that we may establish three varieties of concretions: the first is similar to the form just described, but he terms it exclusively ossification of the fibroid tissue developed in the interior of the valve by inflammation; he calls the second form, ossification of endocardial deposit, on the surface of the valve; and he describes the third as an osseous concretion in a stalactitic form, or as a rough calcareous agglomeration, which constitutes a metamorphosis of the vegetations on the valve. These stalactitic osseous masses, he says, occasion and promote the continued formation of new vegetations; and are consequently very commonly surrounded by them. Calcareous concretions and morbid affections of the valves generally follow the law which determines the great prevalence of disease on the left side of the heart as compared with the right side: ossification, especially, is so rare on the right side that it has been denied altogether. Hasse, however, has seen partial ossification of the pulmonary artery; and Dr. Hodgkins[1] also reports a case of thickening and bony deposit in the pulmonary artery close to the valves.

A condition of the valves remains to be pointed out, which was first demonstrated by Dr. Thurnam;[2] it consists in a saccular dilatation, which he attributes to a gradual distension, and hence terms aneurism of the valves. It is met with in the aortic and tricuspid, but most commonly in the mitral valves. Dr. Peacock[3] has also recorded a similar affection of the valve of the foramen ovale. We find that the dilatation may exist without any lesion of continuity in the tissue; the endocardial lining being traceable throughout the pouch. This, in the case of the mitral valve, projects into the left auricle, and is often filled with a clot of blood. This form would correspond with what is termed true aneurism of the arteries. A second variety is that resulting from inflammation of the valves, by which a solution of continuity is effected in the lining membrane. Rokitansky states that he has found it occur either as a fissure brought on by softening of the membrane, or by disintegration of the subjacent tissue; or again, by ulceration of the endocardium resulting from an abscess proceeding from the lowest part of the valve. Thus, he continues, when the valve has been perforated to

[1] Catalogue of Guy's Hospital Museum. No. 1403.
[2] Medico-Chirurgical Transactions, vol. xix. p. 162, vol. xxi. p. 187, vol. xxiii. p. 323.
[3] Pathological Reports, 1850–51, p. 80. Several instances of valvular aneurism are detailed in the same volume, pp. 72, 77, and 78.

a greater or less extent, the blood which impinges on it penetrates into its parenchyma and causes more or less extensive infiltration. We give this explanation in deference to the authority from whom it emanates;

Fig. 147.



Aneurism of the mitral valve; a pouch projecting into the cavity of the left auricle about three-quarters of an inch high, and half an inch wide. It has burst by an irregular rent on one side. St. Bartholomew's Museum, 12th Series, 62.

we cannot, however, deny that the evidence in favor of the prevailing cause of valvular aneurism being dilatation of the coats, rather than a rupture of the membrane, appears to us to be the stronger. The form of the aneurism is almost invariably that of a circular cup, varying in size from a pea to a walnut; nor does it appear from the cases which we have analyzed, that the affection so uniformly terminates in laceration as Rokitansky affirms.

We have for the sake of convenience reviewed the diseases affecting the individual tissues of the heart separately; but before proceeding further, it may be well to dwell for a brief space upon their complications with one another, and with morbid phenomena in other vital organs. The fact of the intimate connection between a rheumatic diathesis and pericardial and endocardial inflammation, has already been alluded to. We cannot show the relation better than by extracting from Dr. Latham's Lectures on Clinical Medicine, the statistical facts illustrative of the subject, to which that author's large experience had led: The number of cases of acute rheumatism which occurred to him were 136, out of which 90 presented symptoms of heart disease; of these 63 were diagnosed as affecting the endocardium alone, 7 the pericardium alone, and 11 both endo and pericardium. Out of the total number only three proved fatal; they were men, and in them both surfaces of the heart were inflamed. In all cases of heart disease other organs will be liable to be affected in proportion, as different parts of the circulation are more immediately involved. While disorders of the arterial system more directly induce deranged action in the brain, the spleen, and the kidneys; the lungs, the liver, and the chylopoietic viscera suffer chiefly in derangements acting immediately upon the venous system. As a matter of course this distinction is one that cannot be always demonstrated, as in an advanced stage of cardiac disease of either side of the heart, or of any one portion, the entire circulation must of necessity be impaired. On the arterial side we find that more particularly a complication between granular degeneration of the kid-

neys with heart disease obtains; thus, Dr. Bright has shown that in a hundred cases of this disease, the heart presented lesions in at least thirty-five, a number which would probably have been increased if the condition of this organ had been noted with the same care in all. This proportion has been confirmed by the researches of Dr. Taylor.[1] The secondary effects produced by the dislocation of lymph from the left side of the heart, in the brain, the spleen, and the kidneys, by blocking up the arteries, and thus altering the nutrition of the parts to which they lead, we have already alluded to. The influence of valvular disease in producing hypertrophy is a point of great importance, and its connection with pericardial and endocardial inflammation has been especially dwelt upon by Bouillaud. Its influence in affecting the circulation in the brain is undeniable, but it is probable that the frequency with which it induces hemorrhage, either in the lungs or in the brain, has been overrated. In many of the cases on record of cerebral apoplexy connected with cardiac hypertrophy, the result was more justly attributable to the coincident arterial disease than to the increased impulse of an enlarged heart. Pulmonary apoplexy appears rather to be connected with the obstructions to its circulation presented by mitral disease than by an hypertrophic condition of the heart. With regard to the liver, we find that in fatty degeneration of the heart it commonly presents a similar concomitant affection, not to speak of the congestion to which it is almost invariably subject when the return of the blood to the heart is in any way impeded; more than any other organ it is enabled by its size and elasticity, as well as by its functions, to serve as a species of reservoir where the balance of the circulation is disturbed, a reservoir which may be frequently overcharged, but from which we are more able to draw off the surplus without too much debilitating the system than from any other organ. Congestions of the venous system of the entire body are frequent in cardiac disease, and manifest themselves by lividity of the cutaneous surface and of the mucous membranes: and the secondary effects of stasis are shown in these tissues by œdema and hemorrhage, while in the serous cavities they are evidenced by an effusion of serum—one form of passive dropsy. Of the latter, we find the peritoneum chiefly prone to suffer, a circumstance which we may fairly attribute to the absence of any compression, such as we find normally exerted upon all the other serous sacs. With these few remarks on a subject which belongs rather to the domain of the history of disease than the records of morbid anatomy, we pass to the consideration of the conditions with which cyanosis is found associated.

CYANOSIS.

Cyanosis is a term applied to a livid, purplish hue of the cutaneous surface, which is found to accompany some organic and congenital disturbances in the central organ of the circulation, of a more intense character than the slaty tinge which the complexion is very frequently

[1] Medico-Chirurg. Trans., vol. xxviii. p. 536.

observed to assume in acquired disease of the heart. It was formerly attributed, on theoretical grounds, solely to one lesion, a permanent patency of the foramen ovale; and although this occasionally gives rise to the affection, by allowing an intermixture between the blood of the two sides of the organ, and causing it to be circulated through the system, without having undergone the purifying process to which it ought to be subjected in the lungs; it is satisfactorily demonstrated, both that the foramen ovale may remain open, to a considerable degree, throughout life, without inducing any serious disturbance of the circulation; and on the other hand, that various other irregularities in the heart may give rise to cyanosis. Bizot found the foramen ovale more or less open in forty-four out of 155 subjects, in none of whom there was a trace of the morbus cœruleus. Two openings have been found in the ventricular septum, and no cyanosis resulted; a marked instance of this kind in an individual who attained to the age of eight years, was brought before the Pathological Society, by Dr. Quain, in 1847. In such a case, we are justified in assuming that the forces of the two sides of the heart are so exactly balanced as not to disturb the circulation; and the orifice of the pulmonary and systemic arteries being patent, the contents of each side pass into their proper channel. That this is a prevailing law for many cases of cyanosis, is shown by the fact that it frequently does not manifest itself unless there is some further cause for derangement of the circulation, such as a bronchitic affection, to which, it may be remarked, cyanotic individuals are peculiarly subject.

The lesion that appears to be most constantly associated with cyanosis, and which may be regarded as its primary cause, is a contracted state of the pulmonary artery; and, as in that case more than usual pressure will continue to be exerted upon the foramen ovale, this will necessarily remain patulous, and allow a passage of blood from the right to the left auricle; in such a case it may be almost looked upon as a safety valve. Gintrac[1] has analyzed fifty cases of cyanosis, and among them found obstruction at the pulmonic orifice in twenty-six; the proportion is stated to be still greater by other authors. But the blue disease is not necessarily the result of an admixture of the contents of the two sides of the heart; anything causing an arrest in the return of the venous blood to the heart is sufficient to give rise to it. In the first volume of the *Pathological Reports* (1847, p. 25), we find a case of marked cyanosis, recorded by Mr. Ebenezer Smith, in which the foramen ovale was perfectly closed, and had evidently been so for some time before birth; there was no inter-ventricular communication, the pulmonary artery was large, but the left auriculo-ventricular opening was small, and the left ventricle was almost obliterated; the walls were contracted on a small cavity at the base, not exceeding two or three lines in diameter. The aortic opening was also very small, being about two lines wide; and the arch was much smaller than the pulmonary artery. The mitral valve was altogether defective in structure, consisting only of two small bands without any curtains. Here, then, there was an evident arrest at the aortic orifice, which reacted upon the pulmonary circulation, and through

[1] Sur la Cyanose, Paris, 1824.

that upon the systemic capillaries. The lungs were too much charged with blood to perform the duty of aeration effectually, and a congested or cyanotic condition of the surface resulted. Similar instances of the cyanosis being due to contraction at the aortic orifices are on record, but it may also happen without this symptom. Dr. G. A. Rees presented the heart of a child to the Pathological Society, in 1847,[1] in which the aortic was much smaller than the pulmonic orifice, and there was no cyanosis. The ductus arteriosus continuing open, allowed the blood to pass from the pulmonic artery, directly to the aorta, so that the blood distributed to the lower part of the body must have been almost entirely venous.

One of the most palpable instances that has occurred to us, proving how little we are able to account for cyanosis theoretically, was that of a child that lived to the age of nine weeks, and whose heart, after death, was found to present no auriculo-ventricular opening, on the right side, while there was scarcely any inter-ventricular septum at all. Here there had been no cyanosis, although a thorough intermixture of the venous and arterial blood must have necessarily taken place.

Bouillaud[2] is of opinion that the communication between the two sides of the heart, and the consequent admixture of the arterial and venous blood, has, comparatively, little to do with the purple hue of the complexion, which he considers to result, mainly, from the coincident obstacle offered to the circulation by a malformation of the arterial orifices of the heart. The numerous cases on record, in which not only the foramen ovale was patulous, but in which there was further evidence of the actual passage of the blood, directly from one side of the heart to the other, shows, as Dr. Peacock[3] has remarked, that there is a want of just relation between the amount of venous blood entering the general circulation and the degree of cyanosis. The lesions that are found in connection with this symptom, consequently, require to be carefully analyzed, before we can determine the exact part that each bears in its production. They may shortly be enumerated as a patulous condition of the foramen ovale, from the valve not entirely covering the orifice; with this a defective involution of the Eustachian valve is commonly combined: permanent patency of the ductus arteriosus; contraction of the arterial orifices; a deficiency in the intra-ventricular septum; and the malformation in which the aorta springs from both ventricles. The effect upon the heart itself in these cases is to produce hypertrophy and dilatation, more especially of the right ventricle.

Cyanosis is a disease which generally shows itself at or immediately after birth. The circumstance that it occasionally makes its appearance later in life, has induced Meckel and Abernethy to assume that the foramen ovale may reopen, an hypothesis which is unnecessary, as we now know how frequently a communication exists between the auricles, without producing cyanosis, and that this lesion may, under certain circumstances, as in diseased states of the lungs, induce a disturbance in

[1] Reports, 1847–48, p. 203.
[2] Traité Clinique, &c. vol. ii. 690, *seq.*
[3] Pathol. Reports, 1848, p. 202.

the balance of the circulation, sufficient to force the blood through the auricular septum.

Stress has been laid by several authors upon the circumstance that the fingers of cyanotic individuals are found clubbed. We only advert to it, to mention that it is by no means diagnostic of this form of heart-disease, or, in fact, of any distinct malady. A more important point, is an observation that Rokitansky concludes his remarks on the subject with; to the effect that cyanosis is incompatible with tuberculosis, against which he states that it offers a complete protection. We do not deny that this is the prevailing rule, yet it is not as absolute as the author quoted asserts. In the Report of the Pathological Society for 1848 (p. 200), we find a case presented by Dr. Peacock, which refutes the universality of the law. There the post-mortem examination of the individual, a young man, aged twenty, established the following facts: The right lung was extensively permeated by tubercle, and towards the apex exhibited several small cavities; the left lung contained much solid tubercle; the heart was hypertrophic; the pulmonary artery exhibited a complete diaphragm, formed by adhesion of the valves, leaving only a small triangular aperture; the foramen ovale was very widely patulous. There had been cyanosis during life, but not in a very marked degree.

Besides the malformations of which we have just spoken, we find other congenital affections of the central organ of the circulation which are compatible with life, to which we must briefly turn our attention. Those hitherto mentioned have all been instances of an arrest of development; an excess of development is rarely met with in the heart, except as an acquired condition. In all the varieties of congenital arrest, we see a tendency to return to the primitive type of a single pulsating cavity; in itself a sufficient proof, if any be needed, that the organ is not a combination of two originally distinct hearts, but that it arrives at its perfect state by a subdivision of a single cavity. As the growth proceeds through its different stages, from the simplest condition of the pulsating vessel, to the complex mechanism of the perfect heart, we see close resemblances between temporary conditions of the human heart, to permanent conditions of the heart in the lower animals. Thus, the type of the piscine heart is presented in those cases where, in a man, we only find a single auricle and ventricle. Here, an aorta proceeds from the latter, from which the lungs are nourished by the ductus arteriosus, while both venæ cavæ and pulmonary veins discharge into the auricle. Children are known to live several months with this defect, without necessarily presenting any marked symptoms of deranged circulation. In the next degree, we find an analogy with the amphibious heart, the septum ventriculorum being absent, or imperfectly developed, while there are two auricular cavities. A defect in the ventricular septum is commonly associated with that malformation of the aorta, in which it communicates with either side of the heart, the pulmonary artery being displaced, or altogether absent. The defect in the inter-ventricular septum may present various degrees, from a mere rudiment at the apex, to a full development of the partition, with the exception of a minute orifice near the base.

A very curious anomaly, a genuine freak of nature, consists in a

transposition of the pulmonary artery and the aorta, the former arising from the right, the latter from the left ventricle; an accident which is attributable to an abnormal division being effected in the arterial bulb, at the period of intra-uterine life, when the branchial arches are being converted into the arteries of the upper extremities and head, and into the pulmonary arteries. Again, we are informed by Tommasini[1] of an instance occurring in a female, aged twenty-five, who had not been cyanotic until the last days of her life, and had enjoyed general good health, in whom a circular orifice in the parietes of the left ventricle, established a permanent and free communication between this cavity and the pulmonary artery. Other varieties, in the configuration of the heart and the distribution of the vessels, are recorded in the works of Meckel and Geoffroy St. Hilaire, which contain a full account of everything relating to the subject. We have adverted to those most frequently met with. The valves of the heart, in these various malformations, generally present some alteration, being thickened, corrugated, or otherwise changed from their normal constitution. An alteration in the caliber and form of the parietes of the heart is not less frequently found to accompany the arrest of development spoken of; hypertrophy of one or more parts is a very common accompaniment. The valves frequently present a congenital arrest, or excess of development, sometimes independent of any other malformation of the heart, but commonly associated with further lesions. In reference to the arterial orifices, Dr. Peacock[2] observes that the aperture may be defended by a single valve, protruded forwards in the course of the circulation, a condition seen chiefly in the pulmonic artery, or only two valves appear, owing to two having united at their edges; or again, there may be two fully developed semilunar valves, with an abortive valve intervening. Of forty-one cases of defect in the number of the valves, Dr. Peacock found the malformation at the pulmonic, in nine; in thirty-two, at the aortic orifice. Of fifty cases of malformation of the semilunar valves examined by the same observer, nine were examples of excessive development. Of these, he found that eight were cases in which the pulmonic valves were in excess, and in one only was there more than the natural number of valves at the aortic orifice. "In some cases, the excess in the number of valves seems to be due to the division of one of them into two, such divided valves being smaller in size than the others. In others, there are three valves of nearly equal size, with a smaller supplementary valve interposed between two of them. Occasionally, the aperture is provided with four valves, gradually decreasing in size, and in the other cases there may be four valves of nearly equal size, and natural form." Similar defects are met with in the tricuspid and mitral valve, but more rarely.

[1] Quoted by Bouillaud, vol. ii. p. 674, 1841.
[2] Report of the Pathol. Soc., 1852, p. 292.

CHAPTER XXII.

THE BLOODVESSELS.

In estimating the morbid lesions occurring in the bloodvessels, we must bear in mind that they are mere conduits for the fluid, by which nutrition and the metamorphoses of the tissues are effected, and that they therefore bear a very different relation to disease from that presented by the central organ of the circulation, or by the blood itself. It is the more necessary to urge this, as so much that has been written with regard to the main pathological condition, which we are able to excite and observe in the living tissues, inflammation, appears to have originated in the view that the coats of the bloodvessels were the most essential elements in the production and maintenance of the phlogistic process. This is owing to the experiments having been necessarily of a character to irritate the vessels from without, and produce rather a physical than a vital effect. We cannot in this way imitate those constitutional causes of inflammation to which we must generally attribute its production, and in which it is impossible not to recognize the state of the blood as constituting the first element in the production of the phenomena in question. These remarks apply more particularly to the capillaries, but we shall have occasion to see that they also bear upon many of the symptoms met with in the larger vessels. The difference in the direction of the current, in the composition of the blood, in the velocity and force of the circulation, and in the structure of their coats, are points that must not be overlooked in forming an estimate of the diseases of the two great classes of vessels, the arteries and veins. The manifestations of disease in its primary and secondary form are essentially different in the two, as we find their physiological and anatomical relations to be widely apart. The arteries exhibit between their lining membrane and cellular coat a dense fibrous layer, which contains no vessels, and therefore removes the vasa vasorum, which ramify in the cellular coat, much further from the lining membrane than is the case in the veins, nor can any vessels be detected on the lining coat, or between it and the middle tunic. To this circumstance, and not to any difference in the supply of bloodvessels, we must attribute the fact that, while irritation of the lining membrane of the veins rapidly and easily produces inflammatory reaction, it is almost impossible to produce such effects in the arterial lining membrane. Hasse[1] is of opinion that the latter, in both instances, at first merely yields to the alternations of en-

[1] Pathological Anatomy, Syd. Soc. Ed. p. 11.

dosmosis and exosmosis, and does not suffer any organic change until a later period. In arguing on the effects of inflammation in the arteries, we must not forget that the laws of exosmosis and endosmosis apply chiefly to fluids occupying the opposite side of the same membrane. In the arteries, we find deposits chiefly between the middle and lining coat, and of a character to forbid our believing that they can be derived directly from the current circulating in the vessel. Here, then, the exudation from the vasa vasorum has traversed the dense fibrous coat; and there appears to be no reason for refusing to admit that, eventually, the lining coat may be involved in a similar process. Without entering further into these considerations, we may observe that we are unable to join those who look upon the reddening of the internal coat accompanying its thickened condition, which cannot be attributed to post-mortem action, to mere imbibition; but that there must be some change in the nutrition of the parts analogous to inflammation in other parts.

THE ARTERIES.

To proceed systematically, we shall first examine the morbid conditions of the arteries.

We have seen that it is a subject of debate whether the middle and lining coats of the arteries are subject to inflammation; as they possess no bloodvessels of their own, we can scarcely assume them to present symptoms of the primary phenomena of inflammation; but that they may be secondarily involved in inflammatory affections proceeding from the cellular sheath, cannot be doubted. A most interesting case of acute arteritis, in a previously healthy individual, a gentleman, aged twenty-nine, is recorded by Dr. Romberg,[1] where sudden pain manifested itself in the right femoral artery, affecting the distribution of the artery in the limb, then mounting up to the aorta, passed to the left iliac and its branches. Endocarditis followed, and inflammation of the arteries in the left upper extremity; the entire illness lasted from the 20th October, 1844, to the 5th December, of the same year. The post mortem was performed by Professor Froriep thirty hours after death, and the following appearances were found in the arteries: A pale red, firm clot was discovered in the abdominal aorta, close to its division; it blocked up the artery, and adhered closely to its lining membrane, which was smooth and not reddened. This coagulum extended into the two iliac arteries, gradually became thinner, and terminated in a point. At the point at which the left external iliac is given off, there was an equally firm but lighter-colored exudation. The left external iliac, as far as Poupart's ligament, was filled up with a thinner coagulum-containing much cruor; it could be easily detached from the lining membrane, which was thickened, reddened, and friable, and could be easily detached from the fibrous coat. The middle and external coats were also thicker and more friable than in the normal state. Between the membranes

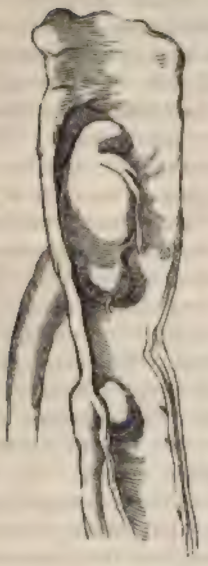
[1] Manual of Nervous Diseases, Sydenham Society's Edition, vol. ii. p. 238. Since the above was written, a very similar case has occurred under the care of Dr. Sibson, at St. Mary's Hospital.

there was an exudation of lymph, which was also distinctly perceptible in the cellular tissue surrounding the arteries. The latter was particularly inflamed under Poupart's ligament, and the neighboring lymphatic glands were tumefied and reddened. The crural artery contained a firm coagulum at the point at which the profunda is given off, which could only be detached with difficulty from the dark red lining membrane, and which extended into the profunda. Further on, the crural artery was filled with a grumous coagulum, and the lining membrane was villous, rough, and much reddened. Then came a free spot, but at the part where it passes through the adductor, it was again closed by a firm coagulum, and the corresponding lining membrane was much reddened, softened, and pulpy. The tissues here were in a state of gangrene, the right internal iliac was unaffected. A firm, pale clot, strongly adhering to the lining coat, was discovered in the external iliac close to the point at which it is given off by the common iliac artery. The crural artery of the same side was narrow and contracted; the lining membrane thrown into folds, containing a solid plug at the site of the profunda; the lining and other membranes being much reddened and thickened. A similar coagulum was found in the left branchial artery at its division, extending into the radial and ulnar. The heart was hypertrophic, and a roundish excrescence was found attached to the mitral valve, which was proved by Professor Müller to consist of fibroid tissue, and to be subjacent to the endocardium. The same author confirmed the fact that a thin layer of plastic exudation matter was found on the arterial coagula, which at many points also invested the lining membrane. For further particulars, and for the author's views on the case, we must refer the reader to Dr. Romberg's work. We have extracted so much of it as refers to the subject under consideration, and because it offers a combination of all those phenomena which writers attribute to acute arteritis, and which are found in the inflammations of other parts of the system as a result of a peculiar derangement of the circulating fluid. In this respect, the case quoted might form an appropriate text for the development of the whole theory of the phlogistic process. Bizot[1] describes as the result of acute inflammation of the arteries, an albuminous exudation of greater or less thickness, of the consistency of jelly, transparent, smooth, sometimes rose-colored, at others colorless, covering the lining membrane. It is occasionally so transparent as to escape attention unless very carefully examined. It occurs in patches, solitary or numerous, and diminishes the caliber of the vessel; in one case, Bizot saw it entirely plugging up the anterior tibial artery. In the aorta, this exudation is formed mostly at the orifice of the arteries arising from the arch, at the mouth of the cœliac, mesenteric, and renal arteries, and at its posterior surface, so as to block up the mouths of the intercostal arteries. An instance of acute inflammation of the aorta is recorded by Mr. Hodgson;[2] it is to this effect: A man was seized with violent pneumonia, which proved fatal in five days; the cadaveric inspection exhibited all the thoracic viscera in the highest

[1] Mémoires de la Société d'Observation, vol. i. p. 311.
[2] On the Arteries, p. 5.

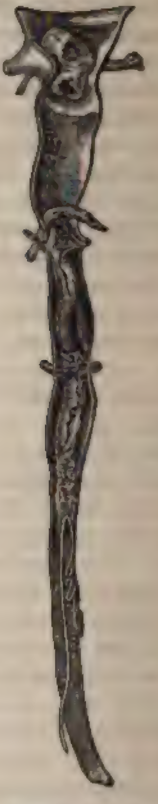
degree of acute inflammation; the aorta was also involved; its internal coat being of a deep red color, and a considerable portion of lymph being effused into the cavity. The effused lymph was very intimately

Fig. 148.



Plastic deposits in aorta.

Fig. 149.



Plastic plugs occluding the axillary artery.

connected with the internal coat of the vessel, and a plug of it had extended into the left subclavian artery, and nearly obliterated the cavity of that vessel. In reference to this subject, some experiments performed by Gendrin[1] are of considerable importance in demonstrating the capability of the coats of the artery giving rise to inflammatory exudation in the strict sense of the word. He found that on injecting an irritant substance into a portion of an artery included between two ligatures, and deprived of blood, a deposit of coagulable lymph took place, which arrested the internal coat, and at last formed a plug filling up the channel. The lining membrane at first was only slightly discolored, and through it a network of injected capillaries might be distinguished on the adherent surface of this tunic to the middle coat. When the

[1] Histoire Anatomique des Inflammations, vol. ii. p. 13.

inflammation had advanced, this was no longer seen, the external coat having become pulpy, rugous, and dull. The suppuration that followed did always coincide with ulceration of the inner coat; the pus, however, was not necessarily deposited in the vessel, but infiltrated into the cellular sheath, forming small abscesses. We may reasonably conclude that in arteritis the morbid products are derived from the vasa vasorum as well as from the contained blood. To sum up: The symptoms of acute inflammation of the arteries are more or less extensive, reddening, softening, thickening, and detachment of the lining coat which exhibits an opaque, plicated condition; the middle coat becomes hypertrophied and friable, and in the external coat we find distinct signs of congestion and exudation. Within the vessel, a coagulation of fibrin and the deposit of coagulable lymph from the blood is seen, and as secondary effects we have to deal with ulceration, laceration of the coats, hemorrhage, and gangrene of the distal parts of the system.

From the time of J. P. Frank,[1] who first drew attention to the subject of arterial inflammation, to the most recent periods, various pathological conditions have been attributed to it; the acute forms have been repeatedly asserted to be the cause of trismus neonatorum, a disease which at present is one of very rare occurrenee among ourselves. Dr. West denies this cause, but Dr. Collis,[2] and recently Dr. Schöller[3] satisfied themselves of its real existence. The latter found inflammation of the umbilical arteries in fifteen out of eighteen cases of trismus neonatorum. There was tumefaction of the umbilicus, reddening and congestion on the external surface; the channel contained pus, and the lining membrane was eroded and invested with an albuminous exudation. Dr. Schöller has carefully examined these parts in all other new-born children who died shortly after birth, and has never succeeded in discovering similar lesions. It does not appear that traumatic tetanus in the adult, to which we may compare trismus neonatorum, is accompanied by similar lesions.

The formation of a coagulum in the artery is a well-known physiological effect of the laceration by mechanical or other means of the lining membrane, and the atrophy or gangrene of the part nourished by the artery is an illustration of the effects following similar obliteration of the channel from disease. We have alluded to the cerebral affections resulting from an arrest in the arterial circulation; senile gangrene is another morbid condition which has been ascribed, by Dupuytren and Cruveilhier, to arteritis. In this there is a marked distinction between inflammation of the two sets of vessels; that phlebitis induces secondary deposits and œdema, while these occurrences are not met with in arteritis. It is even doubted whether the latter ever gives rise to suppuration, but, independently of the cases of suppuration in the umbilical artery quoted from Dr. Schöller, Andral and Hodgson's[4] authority[5] de-

[1] De curandis Hominum Morbis, vol. ii. p. 363.

[2] Dublin Hospital Reports, vol. i. p. 285.

[3] Neue Zeitschrift für Geburtskunde, herausgegeben von Busch, d'Outrepont und Ritgen, vol. v. p. 477.

[4] Anat. Pathologique, tom. ii. p. 379.

[5] On the Arteries, p. 10.

termine the question affirmatively, for these authors state that actual idiopathic suppuration does occur in the artery.

The spontaneous coagulation of the blood in the arteries is not, however, the result of inflammatory action only. It may occur in consequence of a low ataxic condition, which does not permit the vital powers to resist the chemical tendencies that normally ought not to come into play until after death. This spontaneous coagulation is especially met with in the pulmonary arteries, where the occurrence of the inflammatory symptoms has, as yet, not been met with. Mr. Paget,[1] in describing a case of the kind, says, that nearly all the branches beyond the primary divisions of the pulmonary artery contained clots of blood, which, from a comparison with those found in tied arteries, he judged to be from three to ten days old. The clots did not commonly extend continuously from any large branch of the pulmonary artery into many of its successively subordinate divisions, no branch of the pulmonary artery less than half a line in diameter appeared to contain any of these clots, and the pulmonary veins were healthy and empty. The case under consideration proves that a large portion of the pulmonary circulation may be arrested for a considerable period without immediate danger to life, a circumstance explained by Mr. Paget, by assuming a retardation of the circulation in the systemic vessels, in order to allow the quantity traversing them in a given time to be equal to the reduced quantity which in the same time traverses the lungs. In order to keep up the necessary balance, the systemic circulation is as much less rapid than the remaining pulmonary circulation is more rapid than before the obstruction took place.

The formation of a coagulum in the artery does not necessarily block up the entire passage, but may leave a central opening by which the circulation yet continues to be carried on. But after the formation of the clot, it in its turn undergoes various changes; it may become absorbed, or it softens or breaks up into granular matter, and is carried into the capillary circulation, or it is capable of organization, and we then find in it a network of fine bloodvessels. The last point serves to elucidate the observations of the passage of an artery occasionally seen in old coagula formed after the application of a ligature. Lobstein, as we are informed by Hasse, met with an arterial vessel of the caliber of the stylo-mastoid artery running lengthwise through the femoral artery obliterated two years previously by tying. Blandin and Barth have met with analogous instances, to which may be added those cases in which, after the complete obliteration of arteries by ligature, new vessels have been found shooting from their extremities. The general infection of the blood from breaking up of arterial coagula, is a very rare occurrence; a circumstance which establishes a marked distinction between arterial and venous disease; it is referred by Rokitansky to the greater susceptibility of the arterial blood for taking up inflammatory products, which speedily give rise to coagulation and obturation of the vessel, and to the circumstance that their reaction in the arterial

[1] See Mr. Paget on obstructions in the Pulmonary Arteries, Medico-Chirurgical Transact., vol. xxviii. p. 533.

current, being exhausted towards the capillaries in ordinary cases, hinders the general infection of the blood beyond the limits of those vessels.

CHRONIC ARTERITIS.

With regard to chronic arteritis, the opinions of writers are yet more divided than in reference to the acute form. The same difficulties in determining the relation of cause or effect in this subject has been felt by most of the writers on the subject, and have not yet met their complete solution. The older authors attributed the appearances of chronic arteritis to syphilitic taint or mercurial poisoning; some of the more recent, among whom we may mention Corvisart,[1] have held a similar opinion, and Hodgson[2] supports it on the ground that he has observed aneurism, and those organic alterations which generally attend the formation of aneurism, to prevail in subjects that have suffered from venereal disease, and who have taken large quantities of mercury.

While the majority of authors are of opinion that fibrinous deposit, atheroma, ulceration, ossification, and aneurism are the result of a chronic inflammatory process, Hasse absolutely denies its primary existence, and only admits its occasional occurrence as a secondary effect of the degenerative processes alluded to. On the other hand, Rokitansky looks upon chronic inflammation of the arteries as an essential constituent of morbid deposits on the inner coats of the vessel, and its metamorphoses, but is of opinion that it is primarily manifested in the cellular sheath of the arteries, where it produces hypertrophy, thickening, and condensation, followed by a secondary disturbing action upon the normal relation of the inner arterial coats, the fibrous and true lining membrane. It is very certain, however, that we very rarely meet with traces of this superficial cellular inflammation of arteries, except as a result of their implication in morbid processes of the surrounding tissues, as in phlegmonous erysipelas, or in tubercular destruction of the parts; while we constantly have occasion to examine deposits underneath the lining membrane in their incipient stages. Here, we almost uniformly find an accompanying tumefaction and reddening, affecting the entire thickness of the inner coat, which is distinctly perceptible on making a section of the artery. This change is accompanied by a puckering and plicated condition, and extends to a greater or less distance beyond the circumference of the deposit. The reddening, though persistent, is not due to the formation of new vessels; the microscope fails to detect them. The softening, which we find to be a common result of inflammatory action, affects the entire caliber of the artery, and in that case may give rise to a uniform dilatation, or to what has been termed true aneurism. The deposit which invariably takes place in the first instance, between the middle and internal coats, is of a yellowish tinge, and forms slight elevations, in circumscribed dots or patches; as the disease advances, these coalesce, and may thus affect the vessel to a considerable extent. We are not prepared to state, nor are we of opinion, that the chemical

[1] Essai sur les Maladies du Cœur, p. 319. [2] On the Arteries, p. 9.

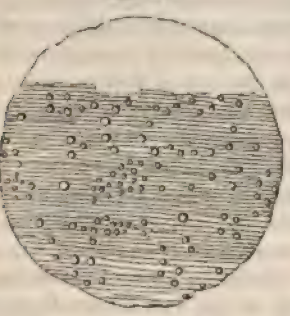
character of the primary deposit is in all cases identical; this would not be in accordance with what we know of the elimination of morbid constituents of the blood. But there is no doubt that in a majority of cases the atheromic deposit is a secondary fatty degeneration of fibrin. We are inclined to suggest that the more purely inflammatory the cause

Fig. 150.

Incipient atheroma and fatty degeneration of an iliac taken from an aged female. The lining membrane is much puckered, owing to the irregular deposit of fibrin between it and the middle coat. A. Naked-eye view of the artery. B. Microscopic appearance of the fibrinous deposit, dotted with oil-molecules. C. A longitudinal section of the artery taken between *—*; *j*, the inner coat with subjacent deposit; *m*, middle coat unaltered; *o*, the external coat.

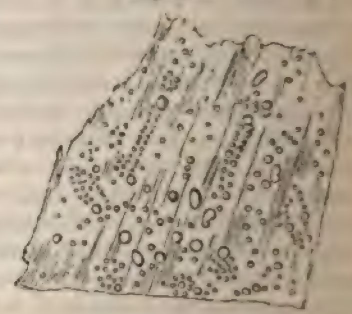
of the disease, the more presumption there is of a fibrinous deposit in the first instance. The fibrin seen by the naked eye presents little or no distinction from actual atheroma; but, on examination by the microscope, we see the delicate fibrillæ characteristic of this matter; and we also detect in it glistening particles of oil, which show the transition to atheroma. The fibrin is itself deposited in distinct laminæ, which may

Fig. 151.



Fatty deposits in internal coat.

Fig. 152.

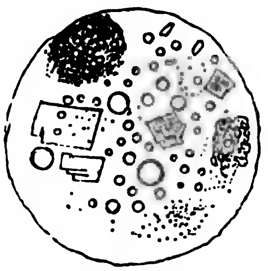


Early stage of atheroma.

easily be separated from one another in the plane of the vascular coats; there is no arrest of the coat investing it on the minor surface of the artery, but this may be peeled off to an indefinite extent beyond the

deposit, showing that in the earlier stage there is no solution of continuity of the lining membrane, a point of some importance in reference to the doctrine expounded by Rokitansky. This author, who treats of the diseases in question as a new formation of the lining membrane from the blood, does not appear to have recognized this form of deposit, which we have delineated, and which is clearly subjacent to the inner coat. Dr. Hope, who does not, however, appear to have confirmed his view by a microscopic examination, looks upon the fibrinous deposit as the first stage of the metamorphosis; and, until further investigations confirm the views of the Viennese Professor, we adhere to the doctrine just adverted to, in preference to what we cannot but regard as a forced explanation. It is, however, but due to the high reputation of Professor Rokitansky, that we should allow him to speak for himself in this question, in order that further investigation may determine the point; he says:[1] "The most frequent form of disease affecting the arteries is an excessive formation and deposition of the lining membrane of the artery, derived from the mass of the blood, and at the same time constituting hypertrophy of this membrane. In a highly developed form of this affection, we find the inner surface of a large artery, as the aorta, covered with a foreign substance, spread over it at separate points or in large patches, and forming a stratum varying in thickness, by which the inner surface of the vessel is commonly rendered uneven; this substance is, in some places, either grayish, grayish-white, faded and translucent, or, in others, milky white, opaque, and similar to coagulated albumen; in some rare instances it is colored by the imbibition of hæmatin, over various extents of surface. Its free surface is at the same time smooth and shining, or dull, and, as it were, wrinkled. It is soft, moist, and succulent in the translucent parts, and dense, dry, tough, and elastic in the more opaque portions; resembling a cartilage or fibro-cartilage, with which it is usually compared, and for which it is still occasionally mistaken. In the latter condition, it adheres internally to the circular fibrous coat." Hasse considers that when chronic arteritis is induced by degenerative processes, we find a deep, dingy, brownish-red color, extending to the middle tunic, a densely injected state of the vasa vasorum of the cellular sheath, and a deposition of plastic material in the caliber of the vessel; these deposits accumulating in masses, adhering to the arterial parietes, and consisting of imbricated layers.

Fig. 153.



Atheroma from old patch.

We occasionally find a general thinning of the coats of an artery without any appreciable change in their composition, in the same way as we also find an hypertrophic condition of the same character. In either case it appears generally to be at the expense of the middle fibrous coat. But, in the great majority of instances, we shall, on careful examination, succeed in detecting some existing, or some ante-

[1] Pathol. Anat. vol. iv. p. 261. Sydenh. Society's Ed.

cedent, morbid deposit to which the condition is attributable. The most important of these is what is commonly called the atheromatous process.

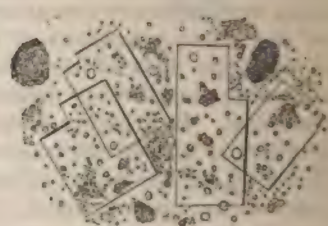
ATHEROMA.

Atheroma,[1] or, as it has also been called by Baillie and others, steatoma of the arteries, is not, as we have already seen, necessarily a soft, pulpy deposit; but appears, in the first instance, as a series of fibrinous layers subjacent to the inner coat of the artery, formed by a process

Fig. 154.

Steatomatous degeneration.

Fig. 155.



Fatty granules, with crystals of cholesterin, from atheromatous deposits in the aorta.—Bennett.

analogous to secretion from the blood. Small oil-globules are early seen in the fibrin, and in the course of time the latter may become completely converted into oil and a fatty crystalline matter, termed cholesterin, which appears in flat, rhomboidal, crystalline laminæ, with sharp outlines; the oil occurs in small globules of a highly refracting character, more or less aggregated in masses, and soluble in ether. In this state it presents a pulpy diffluent condition, and, owing to its yellow color, it may, as it occasionally has, be mistaken for pus. This conversion does not necessarily follow at stated periods, for it is probable that the fibrin may continue unchanged for a long time, until such a tendency prevails in the constitution of the individual of a cachectic or degenerative character, as to give rise to the secondary process. The lining membrane of the artery investing the atheroma, in the first instance, becomes thicker, and assumes a darker color, and is capable of considerable distension by the morbid product. This at first occurs

[1] Atheroma is derived from ἀθήρα, wheat-grits, a pap made of them, hence ἀθήρωμα, an unclassical term, a pulpy substance.

in small points, and, by gradual extension, may occupy surfaces of one or more inches in circumference, and of irregular though definite outline. The liability[1] of the arterial system to be affected, is in a ratio with the proximity to the aorta, this vessel presenting the greatest proclivity. In no disease does the general law of symmetry, which prevails in morbid as well as in physiological growth, manifest itself with so much exactness, as in the one under consideration; the existence of an atheromatous patch in an artery, may lead us safely to infer the presence of an analogous change in its fellow. And this applies equally to those lesions which are consecutive to the primary deposit, as softening and ossification; thus, to quote a single instance from Bizot's memoir, he examined thirty-four cases in which the crural arteries were the seat of patches, and in thirty-three the law of symmetry was observed; in twenty-four cases of consecutive lesion of the same vessel, there was no exception to the law. Even in the aorta we are able, in the incipient stages of the disease, to trace the same law; the patches being found symmetrically placed round the orifices of each pair of intercostal arteries, or forming parallel lines across the main vessel. The two sexes do not differ in any marked manner with regard to the frequency of the occurrence of the patches. The only exception from this is, the well-ascertained proclivity on the part of the female sex to disease of the inferior mesenteric and the abdominal aorta; the ratio of its occurrence in males and females is nearly as one to four. On the other hand, age exerts an undeniable influence in its production; so much so, that, while it is scarcely ever met with before puberty, we rarely open the body of a person in advanced age without finding some arterial lesion of the kind. The liability increases in the exact ratio of the age of the individual, so much so that we can scarcely refuse to look upon the change in a measure as essentially connected with the process of involution. Mr. Hodgson[2] gives two instances of the deposit of calcareous matter in the arteries of infants, but they are extremely rare, and thus only add force to the rule laid down.

OSSIFICATION OF ARTERIES.

The second change which the arterial patches undergo, is that of ossification; a process in which phosphate of lime is deposited in an amorphous condition; it contains no bone-corpuscles or canaliculi, and hence, though the proportion between the animal and earthy matter is about the same as that prevailing in true bone, it is by no means identical with it. As the process advances, the internal arterial coat is generally destroyed, and the mass projects, in the most varied and fantastic forms, into the current of the blood; thus, in its turn, giving rise to further deposits in the shape of fibrinous coagula; or the bony deposit advances under the lining membrane, till it encircles the artery and converts it into a rigid channel, inducing that condition which is found to accom-

[1] See Bizot, Mémoires de la Société d'Observation, vol. i. p. 388.
[2] On the Arteries, p. 23.

pany senile gangrene, fatty degeneration of the heart, cerebral softening, and other morbid processes. The frequency with which different arteries are affected, follows the general rule already laid down, that

Fig. 156.

Calcareous deposition.

Fig. 157.

Annular calcification; it principally occurs in arteries of the third magnitude, such as the popliteal and the femoral. It commences by the deposit of granules of calcareous matter, *a*, which are arranged in lines running transversely to the axis of the vessel; the lines gradually increase in breadth until they coalesce laterally, the intervening spaces being filled up, and the vessel being converted into a rigid tube, *b*.

the liability bears a certain ratio to the proximity of the vessel to the centre of the circulation. This, however, applies only to the systemic circulation; and here, too, there are some marked exceptions, for the cœliac, the hepatic, and the mesenteric, gastric, and hypogastric arteries are scarcely ever found ossified, while the splenic presents this condition very commonly; we must assume that the nature of the contained blood exerts some influence upon the process. Lobstein, who has made out a list showing the comparative frequency of the affection in the different arteries, places the pulmonary arteries last. He also includes branches of the umbilical artery, and the vessels of the placenta. Very small arterial twigs present gritty deposits in their coats; but ossification has not been met with in the capillaries.

One of the secondary effects of atheroma and ossification is, a perforation of the lining coat of the artery, causing, in some instances, an appearance of ulceration; Rokitansky and Gluge deny the existence of

ulceration in the arteries, on the ground that there is no essential analogy between the atheromatous and ulcerous process, and that the deposit itself is not an inflammatory product; but the former author adverts to a peculiar appearance in highly diseased arteries not spoken of by other writers, which he characterizes as minute openings or foramina, interspersed between the deposit, resembling the contracted mouths of small vessels. They lead into canals which penetrate the deposits to various depths, and serve to convey the blood into it and into the circular fibrous coat. The description would lead the reader to suppose that the object of the atheromatous process was to produce this condition, instead of its being a mere result of disintegration.

A remarkable fact in connection with the forms of arterial disease just discussed is, that it appears to confer an immunity from tubercle, while it is closely allied to fatty degeneration; the latter is a point to which attention has been especially directed of late among ourselves; it is one that Rokitansky most pointedly alludes to in the first edition of his great work. There does not appear to be sufficient ground for adopting the doctrine of the older writers, and which Dr. Copland[1] sanctions and enlarges upon, that ossification of the arteries is derived from the same sources as the morbid deposit of stone and gout. The only analogy appears to be that they originate in the blood; but the crasis giving rise to it, as well as the chemical nature of the product, are essentially different. We have already alluded to the circumstances of atheroma inducing aneurism. This disease is one that has long attracted the serious attention of medical practitioners in its advanced stages, in which it becomes not only irksome to the patient, but a source of great danger. Important as the surgical aid is, that in many instances may be rendered, it is easy to understand, from the constitutional character of one of its causes, why operations so frequently fail.

[1] Dictionary of Medicine, vol. i. p. 121.

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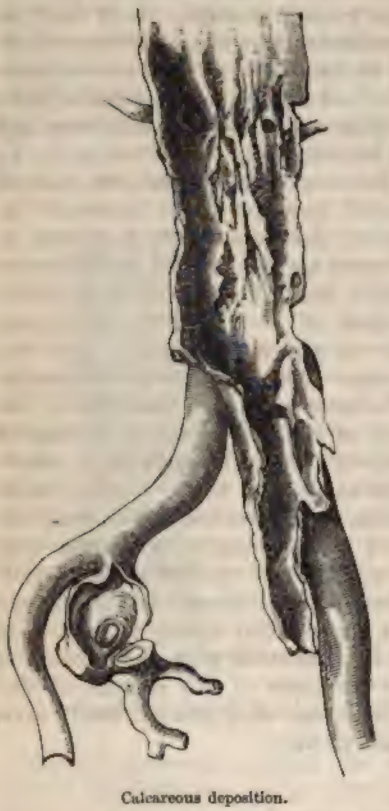
Fig. 156.



Calcareous deposition.

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[1] Dictionary of Medicine, vol. i. p. 121.

CHAPTER XXIII.

ANEURISM.

ANEURISM,[1] or a dilatation of an artery, is connected with two lesions, according to which, from the days of Scarpa and John Bell, downwards, two classes of aneurismatic disease have been adopted by most writers—true and false aneurism—though we shall find grounds for assuming that spontaneous aneurism is in all instances traceable to one ultimate cause—a morbid state of the arterial coats, which may produce accidental varieties in the arterial tumor. The definition of true and false aneurism, ordinarily accepted, is that, in the former, we have to deal with a dilatation, partial or entire, of a certain extent of an artery, without laceration of any of its coats, while in the latter, the dilatation is accompanied by the laceration of one or more of the coats. The distinction is not, however, one based upon a difference in the morbid process; the laceration, except in traumatic aneurism, being an accidental coincidence. The effect of the atheromatous process is to destroy the normal cohesion of the artery, the lining and middle coats are weakened, and the latter may become much atrophied, in addition to being in a degenerative condition. The column of blood acting upon a point thus weakened, necessarily causes the coats to protrude, and the protrusion will correspond in size and extent to the amount of previous disease in the artery. Several small dilatations occurring near one another, may, in the course of time, unite and thus form one large pouch. Dr. Peacock[2] has shown that, in these cases, there is an entire continuity of the lining membrane in the sac, and that, while all three coats may present the normal relation at the orifice, the middle coat becomes so much atrophied as in some cases to disappear altogether at the distal point of the aneurism, so that the internal and external coats come into contact with one another. This is the form which Rokitansky considers identical with the hernial aneurism of authors or with Scarpa's aneurisma spurium. In the simplest form of dilatation the entire caliber of the artery is affected, and the fusiform variety results, or the aneurism is confined to one side of the vessel, and it then assumes the saccular character, with an opening, which is more or less patulous, and may be so constricted as to resemble mere pedicle. It is to the latter that Mr. Hodgson, and some other writers, confine the term aneurism; but there are not sufficient grounds in the nature of the morbid change to sanction this view. Scarpa, following Sennertus, limits the term to those aneurismal tumors which

[1] The term is derived from ἀνευρύνω, I dilate; ἀνεύρυσμα, a dilatation.
[2] Patholog. Reports, 1850, p. 201.

result from rupture of the coats. This is almost invariably found in large aneurismal sacs, and appears to be a main cause of that stratified coagulation of blood met with in them, the clot forming by that law

Fig. 158.

Section of the arch of an aorta, with an aneurism arising from its upper part. The cavity of the sac is nearly filled by laminated coagulum, the internal membrane of the artery is thickened. The sac presses against the trachea, the arteria innominata, and the right carotid and subclavian arteries.—St. Bartholomew's Museum, Series xiii. No. 11.

which enables the human economy to protect itself against dangerous and noxious influences. A solid deposit is less frequently met with in aneurisms formed by simple dilatation of the coats of the vessel.

The form of sacculated aneurisms is generally globular, but they may, partly owing to accidental conditions in the coats of the affected vessels, partly from the pressure exerted by surrounding tissues, assume an oval or more or less irregular outline. Upon the original aneurism, an evolution of secondary dilatations is sometimes met with, and these may even give rise to a further or tertiary multiplication of the disease, so as to induce a sort of mulberry appearance in the tumor; it is the variety to which Cruveilhier has applied the term "anévrisme sous l'aspect d'ampoules à bosselures."

The laceration of the internal coats of the artery may occur at the early stages of the disease, and be the first exciting cause of the aneurismal tumor. The aorta of a lady, whose case is detailed by Mr. Hodgson,[1] illustrated this mode of the formation of aneurism. The coats of the vessel were diseased, and presented at the arch a transverse rent, about an inch in length, which had penetrated to the middle coat. The blood had insinuated itself between the middle and external coats, the latter of which was elevated into a tumor, about two inches in circumference. A similar appearance was found in the body of George II. We see no difficulty in regard to this view of the occasional origin of aneurism. That the early stages should not often be presented to us in

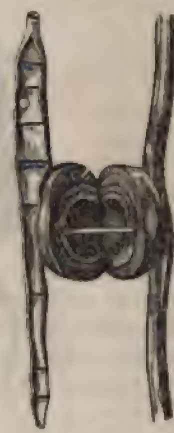
[1] On the Arteries, pp. 39 and 63.

the dead subject, is easily accounted for by the rapid distension that will take place after the first laceration, and the equally rapid laminated deposit of the defensive fibrin. Rokitansky, however, denies this mode

Fig. 159.

Aneurism of the Brachial Artery.

Fig. 160.



Aneurism of the posterior tibial artery, with the nerve spread over the back part of the pouch; the sac is entirely obliterated by concentric layers of fibrin, growing paler towards the surface.—St. George's Museum, F. 41.

of origin altogether, stating that no such rent is ever detected. Another question is, whether laceration of an artery ever occurs without some previous derangement in its coats, in what is termed the traumatic form of aneurism, without a penetrating wound. The extreme pliability and elasticity of the arterial system, compared with all the tissues that surround it, might alone suffice to answer the question; but the direct physiological experiments performed by Mr. Hodgson and Mr. Hunter, and Sir Everard Home,[1] as well as the pathological observations by the former,[2] positively determine the point in the negative. Mr. Hodgson states that he has repeatedly tried, in imitation of Richerand, to produce a laceration of the internal and middle coats of the popliteal artery, by violently extending the leg upon the thigh; but that he has never lacerated the coats of the artery unless the degree of violence was sufficient to rupture the ligaments of the knee, an event which certainly does not generally accompany those accidents to which patients attribute the origin of aneurism.

The contents of aneurismal sacs are fibrinous coagula, which form in successive layers, and accordingly present a concentric arrangement, like the annular rings in perennial plants. The resemblance may be traced still further in the gradual condensation of the outer or external

[1] Transactions of a Society for the Improvement of Medical and Surgical Knowledge, vol. i. p. 144.

[2] On the Arteries, p. 64.

layers, owing to absorption and compression. These also lose their color, and become fawn-colored or white; while towards the interior we continue to recognize the dark color of the blood. The accumulation may proceed to such an extent as to obliterate the cavity, and thus es-

Fig. 161.

Further growth of aneurism prevented by coagulum becoming adherent to the artery around the opening of the sac.—From Hodgson.

Fig. 162.

Spontaneous cure of aneurism of the femoral artery by the sac being filled with coagulum; the vessel remaining pervious.

tablish a spontaneous cure. A considerable amount of organization is observed to take place in some deposits, manifested by the formation of fibres. The existence of a distinct membrane is assumed by most writers to envelop the coagulum. It is a point to which Bizot particularly drew attention, and upon which Hasse dwells forcibly. The latter states that he has never met with an aneurism in which this adventitious membrane was not present. Mr. Bowman has observed that the membrane enveloping the coagula in an aneurism, though apparently of exactly the same nature as that lining the arteries, differs from it in not presenting any epithelium. We would not lay any stress upon this distinction, inasmuch as the presence of epithelium in a healthy artery is often, to say the least, extremely doubtful. The coagula form in proportion as the sac is cut off from the rest of the circulation, the more shallow it is, and therefore the more exposed to the force of the current, the less the liability to the formation of fibrinous laminæ, and the less, we may also add, the coincident danger of perforation of the coats of the vessel. It is through the agency of this deposit that a spontaneous cure may take place, either by an obliteration of the sac, or by pressure upon the artery, and consequent obliteration of its channel. The coagula may subsequently undergo secondary metamorphoses, such as a conversion into

cholesterin, or cretaceous matter. But it does not always form, and we may meet with a series of aneurisms on the same vessel, some of which present the deposit, while others are empty. In number and size there is a great diversity, as also with regard to the liability of different arteries to be affected, as well as in respect of sex. The following table contains an analysis of sixty-three cases, examined by Mr. Hodgson, which illustrates the last two points. It shows at once the great liability of the male sex, and the prevailing tendency of certain arteries to be affected:—[1]

	Males.	Females.	Total.
Ascending aorta, innominata, and arch of the aorta	16	5	21
Femoral and popliteal	14	1	15
Inguinal	12	0	12
Descending aorta	7	1	8
Subclavian and axillary	5	0	5
Carotid	2	0	2
	56	7	63

Bizot's statistics agree closely with those of Mr. Hodgson as to the different liability of the two sexes; out of one hundred and eighty-nine analyzed by him, one hundred and seventy-one occurred in men, and eighteen in women, which is even more in favor of the latter than Mr. Hodgson's table. The proclivity to aneurism is also determined by the age of the individual; it is unknown to childhood; the greatest tendency to the disease exists at the middle period of life, as shown by the following analysis of one hundred and eight cases:—

From 10 to 19 years	1 subject
" 20 " 29 "	15 subjects
" 30 " 39 "	35 "
" 40 " 49 "	41 "
" 50 " 59 "	14 "
" 60 " 69 "	8 "
" 70 " 79 "	2 "
" 80 " 89 "	2 "

As an aneurism enlarges it necessarily displaces the adjoining tissues, and causes an absorption of those that offer any resistance. The danger of an aneurismal tumor depends upon its site, and upon its vicinity to vital organs whose functions are liable to be interfered with by pressure. It is thus that aneurism occurring in the thorax and in the regions of the neck threatens life, before the arterial disease has put on any dangerous appearance, by narrowing the trachea, by compressing the œsophagus or other vessels. The extent to which absorption prepares a passage to an advancing aneurism is in some cases extraordinary; an aortic aneurism by this process may pass through the thorax or eat into the vertebral column. In these cases, as Rokitansky describes, not only the bone is destroyed but the aneurismal sac itself becomes fused with the periosteum, and the other fibrous structures that usually invest the bones. In this way the exposed vertebral column

[1] The table is essentially the same as that given by Mr. Hodgson, but differently arranged. It excludes aneurisms arising from wounded arteries, and aneurisms by anastomosis.

may constitute a portion of the aneurismal wall. Hodgson[1] has pointed out, that, as the aneurism advances to the surface of the body, it induces

Fig. 163.

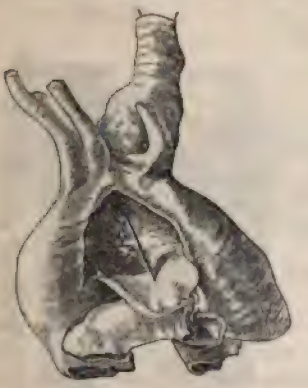


Fig. 164.

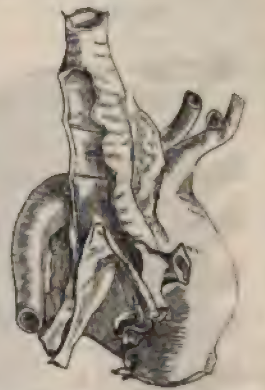


Fig. 163 exhibits a front, and Fig. 164 a back view of an aneurism of the arch of the aorta, which burst into the trachea. The opening into the aneurism from the artery, and the atheromatous patches between the coats of the latter, are well shown.

sloughing of the integuments, and an eschar forming on the tumor itself, its discharge gives rise to fatal hemorrhage; the same is the case when

Fig. 165.

Aneurism of the aorta, which induced caries of the vertebræ, and fatal compression of the spinal cord.

the aneurism opens into a cavity lined with mucous membrane. But a different result takes place when the sac projects into a serous cavity: in this case the membranes do not slough, but the parietes of the tumor become softened and thinned, and a laceration is effected. If a rupture

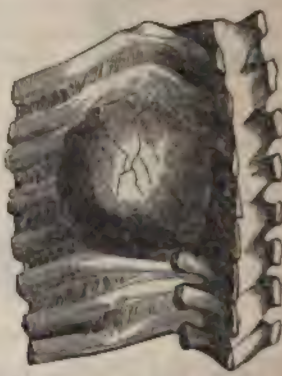
[1] The Diseases of the Arteries, &c. p. 85.

of the internal and middle coats alone takes place, the external coat remaining entire, the blood may separate the latter to a greater or less extent without forming a sac; it then causes what has been termed, by

Fig. 166.

Front view of aneurism of aorta.

Fig. 167.



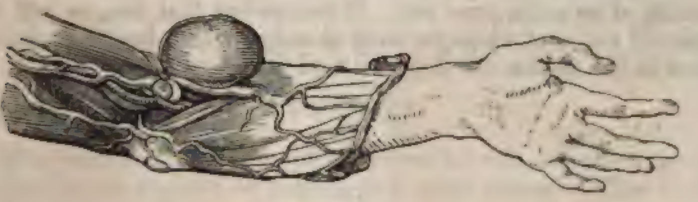
Back view of same preparation, showing the aneurism, producing absorption of the ribs, and making its way to the surface. Death was caused by part of the coagulum falling into the artery.

Laennec, the dissecting aneurism. We sometimes meet with small ecchymoses under the lining membrane of the aorta in the dead body, which indicate the commencement of this form of aneurism. A minute and sometimes imperceptible fissure in the inner coat allows of the permeation of a small quantity of blood, and the first step having occurred, a succession of similar deposits may soon cause a greater accumulation, and necessarily a coincident separation of the coats. Nothing has been added by later writers to the observations of Mr. Hodgson on the subject of the spontaneous cure of aneurism; we cannot do better than to extract his own terse summary of the subject; first, the whole tumor may be removed by sphacelation, in consequence of extreme inflammation excited by the distension of the surrounding parts; secondly, the tumor, as we have already had occasion to observe, may assume such a position as to obliterate, by its pressure, the superior or inferior portion of the artery communicating with the sac; and thirdly, the gradual deposition of fibrin in the sac and the artery leading to it, may render them impervious, and allow a subsequent process by which the tumor is removed. In the latter cases a gradual absorption of its contents takes place, the tumor becomes harder and smaller, and the establishment of a collateral circulation restores the balance of the circulation.

Before quitting the subject of aneurism, we must allude to certain peculiarities in connection with its occurrence in different parts of the arterial system. We have seen that aneurisms are almost limited to arteries of the largest size; in smaller arteries, as in the radial and ulnar or tibials, they are rarely met with. They are altogether extremely rare in the upper extremity; they here almost invariably arise

from carelessness in venesection, especially if, as on the continent, a spring-lancet is employed. In such a case the result generally is a communication between the brachial artery and a cubital vein, especially the basilic, forming what is called varicose aneurism. The

Fig. 168.



From Liston.

smallest arteries in which spontaneous aneurism is met with are the coronary of the heart and the cerebral. An instance of the former, which is extremely rare, is reported in the Records of the Pathological Society for 1848; it was discovered by Dr. Peacock in a man aged fifty-one, who had presented no symptoms of cardiac disease before death. The tumor occupied the left coronary artery, and was about the size of a pigeon's egg, containing lacerated coagula, which were intimately adherent to the lining membrane. There was some atheroma in the aorta. Aneurism of the cerebral arteries, though not common, has of late been shown to be more frequent than was at one time supposed. It is generally seated at or near some part of the circle of Willis; it may attain the size of a walnut, and more, though it is commonly smaller; it is met with chiefly between the age of forty and fifty. Here, too, the male sex presents a much greater liability than the female; showing that the former manifestly possess a marked aneurismal diathesis, and that the increased tendency is not due to accidental circumstances. We find two good instances of aneurism of the cerebral arteries in the Reports of the Pathological Society, presented by Dr. Hare[1] and Dr. Roe;[2] the one in the left posterior communicating artery, the other in the anterior cerebral; this one of unusual size, being as large as a hen's egg, had caused partial absorption of the sphenoid bone upon which it rested, and a flattening of the adjoining portions of the brain. Both the cases alluded to occurred in females; and Dr. Roe's case was still further remarkable from its affecting the patient at the early age of twenty-one. Dr. Brinton,[3] from an analysis of about forty well-authenticated cases of cerebral aneurism, finds that three-eighths terminate in rupture, one-eighth from simple loss of functions by pressure, one-eighth by convulsive attacks, one-eighth by congestion or hemorrhage of the brain, one-eighth by inflammatory conditions of the brain, and one-eighth by coincident disorders or accidents. In three instances Dr. Brinton found the aneurisms more than one in number; in one instance three were found; in one, the opposite carotids were symmetrically affected.

[1] Report, 1849–50, p. 169. [2] *Ibid.*, 1850–51, p. 46. [3] *Ibid.*, p. 48.

The pulmonary artery and its distributions are not subject to aneurism, beyond an occasional dilatation of the orifice adjoining the heart. The mechanical injury of an artery may cause the effusion of blood into the surrounding parts, which constitutes what has been termed diffuse false aneurism. It generally produces gangrene; but inflammatory re-action may be set up, and establish definite limits, and thus lead to the formation of an aneurismal sac. It is manifest that this, as well as the aneurismal varix, of which we have already spoken, has no pathological relation to the disease of which we have been treating.

CHAPTER XXIV.

THE VEINS.

THE physiological relations of the veins, as well as their anatomical structure, would lead us to assume that their diseases differ in many points from those affecting the arterial system. The absence of an elastic coat, their greater collective capacity, the direction of the contained current, their frequent superficial position in the body, the nature of their contents, and the absorbent power of these vessels, are elements which influence their morbid states materially. Some of the most virulent manifestations of disease that we are acquainted with are symptoms of diseases of the veins, and it is most probable that in the great majority of cases of poisoning, whether the toxic agent be introduced by the stomach, by the skin, or by inhalation, they are the medium by which it is conveyed to the central organs and prostrates the vital energies. It is in the study of this subject that the value of pathological inquiry has been particularly demonstrated. Hunter was the first to open the way, and since that period the scalpel has shown that many previously unintelligible malignant conditions are attributable to phlebitis.

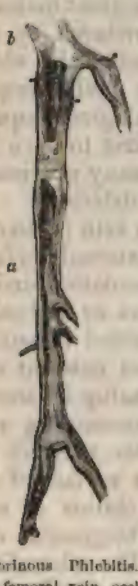
Phlebitis may be acute or chronic. Inflammation of a vein is characterized by a reddening of all the coats, owing, on the external surface, to capillary congestion in the lining membrane, to a transudation similar to that found in the coats of the arteries. It is only when we find other concomitant symptoms of inflammation, that we are justified in setting the latter down to that cause. According to Lebert,[1] the nutrient vessels of the veins, which in the normal state are scanty, during inflammation become excessively developed, forming large anastomosing networks. As the inflammation advances, a second vascular network extends from the former, and is described as passing to the surface of the lining membrane of the veins. Lebert states this evolution of new vessels to be identical with what is perceived in other phlegmasiæ, and to be a uniform accompaniment of incipient phlebitis. The color of the lining membrane fades at the margin of the inflamed portion into the pale, healthy tissue, and itself presents various subsequent modifications of tint, becoming more or less mottled, violet, or fawn-colored. An exudation of serum is next perceived in the coats of the vein; they become thickened, so that when cut across the vessel may remain patulous like an artery. A deposit of fibrin in the channel of the vein follows, and it will depend upon the character of the deposit, and this in its turn upon the constitution of the individual, whether it suffices to

[1] Physiologie Pathologique, vol. i. p. 272.

arrest the disease and render it a mere local affection, or whether it becomes a fresh source of contamination. The more intense the phlogistic process, the more thoroughly we shall find it affecting the coats of the vessel, so that the latter, as Gendrin observes, may appear to form a dark red cord surrounded by and closely adherent to a friable, red cellular sheath filled with bloody serosity, and intimately attached to the surrounding tissues. Gendrin has demonstrated that the lining membrane of veins possesses the same power of exuding lymph into their cavity as that of arteries; we must not, therefore, look upon the coagula found in the veins in phlebitis as resulting solely from a chemical elimination of the contained blood. Lebert describes the adhesive fibrinous exudation as being effected by the new-formed capillaries between the coats, but he regards it as the rarer issue of phlebitis, while he attributes its very fatal character to the prevalent suppurative character of the disease.

It is probable that the first effect of phlebitis is to cause the formation of a layer of exudation matter on the lining membrane, and that this in its turn gives rise to the deposit of further coagula from the blood; the concentric lamination which may generally be traced in the plug, and the firmer connection between it and the vessel in phlebitis, than in those cases of spontaneous coagulation of the blood from other causes, confirm this view. The coagulum itself may extend considerably beyond the primary seat of inflammation, and it terminates in a conical point; the plug generally reaches as far as the next main vessel with which the inflamed vein communicates, while the smaller branches, which contribute to its formation, become choked with lymph. A cure may take place at this stage, either by a permanent obliteration and contraction of the vein, as we see in favorable cases of operations for varicose veins, or by a disappearance of the coagulum and a restoration of the vessel to its normal condition. Hasse was fortunate enough to experience this event in his own person, after the whole system of the saphena up to its junction with the crural vein had become blocked up; he attributes the dispersion of the lymph not so much to absorption as to its resolution and liquefaction, an opinion which is rendered probable by the position of the deposit as well as by the absence of chemical changes which render it hostile to the vital powers.

Fig. 169.



Fibrinous Phlebitis. *a.* The femoral vein, occluded by solidified contents. At *b*, the saphena enters; and consolidation ends abruptly there.

This, however, is not the usual termination of phlebitis, which more commonly leads to a suppuration of the clot, and secondary phenomena of the most destructive character, resulting from this process. In this case, a soft, straw-colored spot forms in the centre of the coagulum, the lamination of the latter disappears, until the whole is converted into a grumous mass. When portions of this mass are pro-

pelled into the circulation, the symptoms of poisoning result, and we find the individual particles of the morbid product occasioning coagulation of the blood, or the formation of abscesses at distant points. Coagula resulting from this cause are most commonly met with in the right side of the heart, and in the distribution of the pulmonary arteries. They are laminated, and are softened in the interior, where there may be a nucleus of pus. The introduction of pus into the circulation is generally accompanied with symptoms of a general prostration of the powers, of the most intense character, which are due rather to the infection of the blood, or pyæmia, than to the local results in the shape of lobular abscesses, or secondary purulent deposits in distant viscera. Cruveilhier is of opinion that, after the occurrence of suppuration, what he terms a sequestration of the pus may take place, and prevent the general infection, by the formation of fresh coagula in advance of the point of suppuration, in consequence of which the pus may be absorbed or be discharged externally, in the shape of an abscess.

Authors of our own country were the first to trace the connection between secondary deposits and local injuries. Morgagni and Desault had particularly alluded to abscesses in the liver following cerebral lesions, but it was reserved for Arnott[1] and Davis[2] to show the actual physical connection between these occurrences, and to establish the real nature of phlebitis. The latter was the first to demonstrate that it is the pathological phenomenon constituting phlegmasia alba dolens, a malady accompanying the puerperal state, which had previously been attributed to a reflux of the lochia, to milk depôts, or to obstruction of the lymphatics. He showed, by a post-mortem examination of four fatal cases, that it resulted from inflammation of one or more of the principal veins within, and in the immediate neighborhood of the pelvis, producing an increased thickness in their coats, the formation of false membranes on their internal surface, a gradual coagulation of their contents, and occasionally a destructive suppuration of their whole texture. Dr. Graves,[3] though he admits that the veins are involved in this disease, looks upon their inflammation as secondary to a general inflammation of the tissues, produced by a morbid impression made on the ultimate ramifications of the sentient nerves of the extremity; on the other hand, Gendrin considers the swelling accompanying phlegmasia dolens to be a mere accidental concomitant of the phlebitis. Many cases of puerperal fever are mainly due to phlebitis of the uterus, and secondary inflammations induced by the former. This appears to have been more particularly the case with the frightful endemic of puerperal fever, which used, until very recently, to rage in the great hospital of Vienna. Dr. Semelweiss made the discovery that it mainly was due to the introduction of poisonous matter into the vagina and uterus of parturient women, by the medical men and students, who had recently been handling post-mortem specimens. The proper precautions that have since been adopted have almost banished the disease.[4] Thus, while in

[1] Medico-Chir. Trans., vol. xv. p. 1.

[2] *Ibid.*, vol. xii. p. 419.

[3] Clinical Lectures, &c., vol. ii. p. 290.

[4] See Dr. Routh on the Endemic Puerperal Fever at Vienna, Med-Chir. Trans., vol. xxxii. p. 27.

1846 there were 459 deaths among 3354 females, in 1848, after the employment of chlorinated solutions by the medical attendants for their own purification, had been introduced, the deaths in 3356 patients had sunk to the comparatively small number of 45. In many cases of phlebitis resulting from venesection, the effect has been traced to a similar cause, viz: the introduction of poisonous matter into the vessel, either by the lancet, or by the use of foul sponges. It was from its occurrence after phlebotomy that the attention of John Hunter was first drawn to the subject.

For a long time the liver was supposed to be the only organ in which metastatic abscesses, as secondary deposits used to be called, were found, and then only in connection with injuries of the brain. It was first shown by Arnott that no organ of the body is exempt from this lesion, though it occurs more frequently in the liver and the lungs, and next in order in the kidneys, the spleen, the heart, and superficial tissues, while it is met with but rarely in the brain and the cavities of the eye. The abscesses vary in number, but when found in one organ, we may expect to find them in others also, and it is rare to meet with a solitary secondary deposit. Thus, in a case that occurred at St. Mary's Hospital, in a boy, in whom crural phlebitis was brought on by an accidental contusion, and death occurred within six days of the injury, and three of the occurrence of any alarming symptoms, secondary deposits were found in the lungs, the heart, and the kidneys. Velpeau[1] relates the case of an individual, in whom, from fifteen to twenty abscesses were counted in the brain, from eight to ten in the lung, and purulent deposits were also found in the kidneys, the spleen, and the liver.

We find the law of symmetry prevailing in the present as in other diseases; thus in the viscera, as well as in the superficial tissues, abscesses, resulting from phlebitis, are constantly met with, at points most closely corresponding with one another on the two sides of the body.

The introduction of a deleterious agent from without is not the sole cause of phlebitis, nor is absorption of pus necessarily followed by the serious consequences to which we have adverted; we must, therefore, assume, that in those cases in which it occurs, there is a predisposition determined by a cachetic condition in the blood, to which it is attributable. Rokitansky treats of a definite form of phlebitis, depending on coagulation of the blood, in which the coagulation within the vessel is the primary phenomenon, whilst the inflammation of its coats is merely a secondary affection. When the coagulum is once formed, which may take place at different points and at different distances from the centres of infection, the inflammation of the coats follows. To this class we must refer cases of phlebitis recorded as having supervened upon inveterate lues, or varioloid disease,[2] or upon catarrhal or rheumatic affections.

In addition to the cases of inflammation of individual veins, already treated of, there are others to which it is right that we should especially advert. An inflammation of the umbilical vein in infants is mentioned by Kiwisch,[3] as occurring almost epidemically; and Dr. Lee has also

[1] Revue Médic. vol. x. p. 442, 1826.
[2] See Puchelt, das Venensystem, vol. ii. 88, 1843.
[3] Die Krankheiten der Wöchnerinnen, Prag. 1840, vol. i. p. 112.

met with it coincidently with the epidemic occurrence of metro-phlebitis. The affection generally commences between the second and fourth days, and is followed by peritonitis and icterus. In the adult, we meet with inflammation of the vena portæ, which may occur idiopathically, or by extension of inflammation from the mesenteric veins. It has been seen resulting from a fish-bone penetrating through the coats of the stomach, into the superior mesenteric; and the cases in which no such lesion could be discovered have been set down to metastatic irritation, to the irritation produced by spirituous beverages, suppressed hæmorrhoids, gout, or erysipelas. The occurrence is marked by the appearance of what exactly resembles the formation of numerous abscesses in the substance of the liver, but which, on close examination, prove to be accumulations of pus in the branches of the vena portæ. The cases of this disease that are recorded are so few that we are induced to quote the following instance, which fell under our own observation; it is the more remarkable as it presents the only instance of apparently idiopathic ulceration of the trunk of the vena portæ, that has been published. It occurred in John Wright,[1] a laboring man, a patient of Dr. Alderson's, at St. Mary's Hospital, who, seventeen days previous to admission, was seized with a shivering fit; since then he had suffered from rigor, followed by heats and perspirations at irregular intervals. On the 1st of October, 1852, a fortnight after admission, the skin is first reported to have been somewhat jaundiced, the pulse eighty-eight, tongue coated, loss of appetite, a burning sensation at the top of the sternum, with great depression of spirits. The yellow tinge of the skin continued, and the fits were fewer in number. On the 11th, the dulness of the hepatic region was found increased, and the stools are noted to have been dark. On the 18th, the skin was less yellow, the percussion of stomach and colon was tympanitic—no pain—no increase in the hepatic

Fig. 170.

Section of liver exhibiting the appearances presented in inflammation of the Vena Portæ.

dulness. The shivering fits now returned more frequently, pleuritic symptoms supervened, the patient became more and more weak, more jaundiced, and drowsy, and sank on the 24th of October, 1852. In the

[1] The case is abridged from the Records of St. Mary's Hospital.

thorax, the deposit of fresh lymph on the lower edge of the right lung, was all the evidence of recent disease to be found. The lungs, the heart, are noted as being healthy, so also the kidneys and the brain. The liver was found much enlarged and dark colored, feeling soft at many points; on removing it from the body, about two or three ounces of purulent matter escaped from the portal vein; the organ, at the same time, shrinking under the hand. On incising the liver, numerous bright yellow circumscribed spots appeared, closely resembling abscesses: they varied in size from a pin's head to a walnut. They proved to be all connected, occupying the ramifications of the portal vein, gorged with pus, of a perfectly laudable appearance. On examining the portal vein, it presented close to the point at which the spleen and superior mesenteric met, a puckered, ulcerated appearance of its inner surface, extending for about an inch towards the liver; the ulceration was found to have penetrated through the inner coat, the edge of which was turned up, and well defined towards the healthy part of the vein. The ducts and the hepatic vein were found healthy, as also the orifices of the mesenteric and splenic veins. The surface of some of the portal branches was smooth, and these only seemed implicated as receptacles for the pus, while, in others, the lining membrane was destroyed, and a pyogenic layer substituted. The hepatic cells proved everywhere natural, the lobules were loaded with yellow pigment in the middle, and there was marked hepatic venous congestion, which it may be observed, closely resembled congestion of the inter-lobular plexuses. Mr. Blyth, who analyzed the organ, found it to contain neither sugar nor cholic acid, nor did the bile obtained from the gall-bladder contain either of these constituents; it follows, that the pigment in the hepatic cells was not biliary matter. The intestines were only partially examined, but so far appeared healthy. This case contradicts the assertion of Rokitansky, that inflammation of the vena portæ invariably induces purulent and ichorous abscesses in the liver, and abscesses in the lung, with a very highly developed pyæmia. No abscess was discovered either in the hepatic or pulmonary tissue, nor was there any evidence of pyæmia; the suppuration was limited, in the most remarkable manner, to the trunk and branches of the vena portæ.

In the cranium, we meet with inflammation and suppuration of the sinuses of the dura mater as a consequence of direct injury, and not unfrequently as a result of otorrhœa and caries of the fibrous portion of mastoid cells of the temporal bone. Cerebral phlebitis is necessarily commonly associated with meningeal inflammation. It appears that the sinuses of the dura mater are liable to a chronic form of inflammation in children, leading to their obliteration and conversion into fibrous cords. Such a condition has been found by Tonnellé, and by Gintrac, in cases marked by symptoms of cerebral congestion and apoplexy. The latter author[1] gives the following case in illustration of this condition: a child, aged four years, was liable from its birth to a temporary suspension of voluntary movement. There were no premonitory symptoms, and the

[1] Recueil d'Observations, Bordeaux, 1830. Quoted by Andral, Clinique Médicale, tom. v. p. 266.

attack occurred equally in the erect and recumbent position; the intellectual faculties were maintained, but the power of articulation was suspended. The child died of pneumonia, supervening upon variola. The post-mortem examination exhibited the superior longitudinal sinus converted into a hard cord, the veins in connection with which were filled with coagulated blood. The walls of the sinus were thickened; dense, and of a yellowish color; it contained a solid clot; no further lesion was discovered in the cranium.

Among the local cases of phlebitis not followed by general infection of the blood, those of the hæmorrhoidal veins are the most frequent, though it is a complication much to be feared in all operative proceedings directed to their cure, as well as to that of varicose veins of the legs or spermatic cord.

Rupture of the large veins is an event very rarely met with; Haller[1] quotes a case of rupture of the vena cava inferior, attributed to eating ice; but most of the instances recorded[2] were brought on by mechanical injury. The rupture of smaller veins often occurs as a result of sudden and forced distension. Thus, small ecchymoses are frequently brought on in the conjunctiva by violent coughing. The veins of the lower extremities have been found ruptured by spasm of the muscles of the calves. The hemorrhage that constitutes epistaxis, the menstrual and hæmorrhoidal discharges, is rather analogous to the process of exosmosis than a result of actual rupture, and therefore is rather more immediately connected with the capillary than the venous circulation. A spontaneous rupture of larger veins occasionally results as a secondary consequence of varices. The sanguineous tumors in the labia of pregnant and parturient women, are attributable to this cause, as also the laceration of varicose veins of the extremities, with or without coincident ulceration.

DILATATION OF VEINS.

Dilatation or varicosity of the veins is a subject which has attracted the attention of pathologists from the days of Hippocrates, who already distinguished between two kinds, which he termed hæmorrhois and cirsus.

The affection is also treated of by writers under the generic term of phlebectasis. It consists mainly in an enlargement of the caliber of the vessels, and may or may not be accompanied by an alteration in their coats. Briquet[3] avails himself of these differences for establishing his classification. He assumes three varieties—simple dilatation; uniform dilatation, accompanied by thickening of the coats; and irregular dilatation, with thickening or attenuation. The distension is generally owing to some impediment being offered to the return of the blood to the heart; and we therefore most commonly meet with it in parts in which the surrounding tissues are lax, and consequently do not offer a sufficient

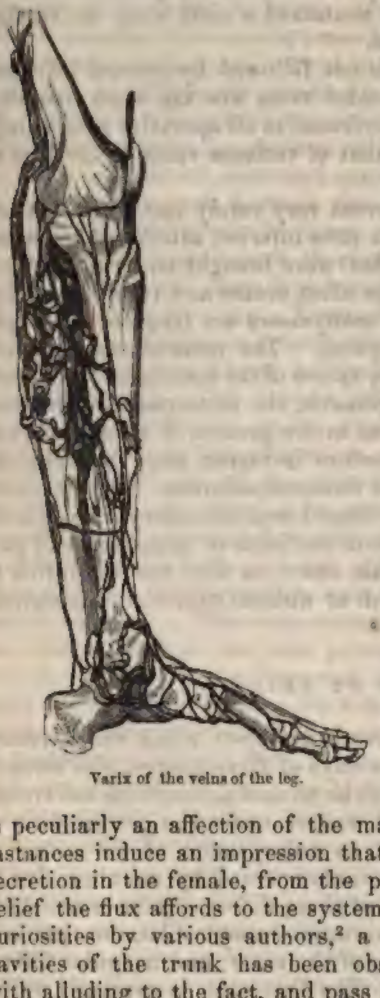
[1] Elementa Physiol. vol. i. p. 130.

[2] James Kennedy has collected all the known cases of rupture of the vena cava inferior, in London Medical Repository, vol. xx. 1823.

[3] Histoire des Inflammations, vol. ii. p. 9, *seq.*

resistance to the pressure of the blood. Hence, varicose veins are most commonly met with in the vicinity of the rectum and pudenda, and in the lower extremities. The veins swell, and assume a nodulated appearance and tortuous course, while the increased local pressure gives rise to an hypertrophy of the coats. Gendrin[1] and Briquet attribute the latter to chronic inflammation. A necessary consequence of the dilatation is an insufficiency of the valves, which no longer close the passage to the regurgitating current. They suffer a solution of continuity, and may become partially or wholly obliterated. The occurrence of phlebectasis is connected with a peculiar constitution, which Hasse terms a morbid predominance of the venous system, a venous habit of the body, which may be characterized as one of general laxity of fibre and want of bone, associated with a tendency to local congestions. Age exercises a marked influence upon the occurrence and prevalence of the affection. It rarely manifests itself in any form until puberty, and is most common during the prime of life, as the tendency to it gradually ceases with advancing years. An hereditary predisposition may very frequently be traced. There is also a marked difference in the two sexes in regard to their proclivity to certain forms of the disease. Thus, the hæmorrhoidal form is peculiarly an affection of the male sex, and its symptoms in many instances induce an impression that it is an analogue to the menstrual secretion in the female, from the periodicity of its occurrence, and the relief the flux affords to the system. In some rare cases, recorded as curiosities by various authors,[2] a dilatation of the large veins in the cavities of the trunk has been observed. We must content ourselves with alluding to the fact, and pass to the consideration of the ordinary forms of the disease.

Fig. 171.



Varix of the veins of the leg.

Varicocele, or cirsocele, affects the male sex commonly at the commencement of puberty. It consists in a dilatation of the veins of the spermatic cord, and prevails more on the left than the right side—a circumstance attributed to the more circuitous route taken by the left

[1] Archives Générales de Médecine, vol. vii. p. 200 and 396.
[2] See Puchelt das Venensystem, vol. ii. p. 378, *et seq.*

than the right spermatic vein. How rarely it affects the right side is shown by the fact that, in one hundred and twenty cases operated upon by Breschet, all but one occurred on the left. It is important, on account of the atrophy of the testicle, which it is likely to induce, from its causing hæmatocle, by hemorrhage into the tunica vaginalis, and from the influence which, in common with all sexual diseases, it exerts on the mind of the patient. The form of varicosity in the female sex, corresponding to varicocele in man, is enlargement of the vaginal and pudendal veins, which, especially during the advanced periods of pregnancy, are the cause of much suffering, and may, during labor, give rise to very severe hemorrhage.

Hæmorrhoids, or piles, consist in an enlargement and varicose condition of the veins surrounding the anus, and may occur in terminal branches of the inferior mesenteric, a tributary of the portal vein, or of the internal iliac. They protrude in the form of bluish nodes, or form flat sessile tumors. From their position, and the frequent pressure and congestion they are subject to when once formed, they are liable to inflammatory attacks; in consequence of which the surrounding cellular tissue condenses and hardens. Small cysts are formed in the latter, into which blood is effused, and they then exhibit a complex structure, which has been the source of much disputation. Abernethy and Kirby have even gone so far as to deny that they were owing to varicosity of the veins, and have asserted them to be mere sacculated prolongations of the condensed submucous tissue. The sequelæ to which they give rise are hemorrhage, ulceration, and prolapsus of the rectum. The periodical character that is often observed in the sanguineous flow, is one that peculiarly deserves the attention of the physician. In man the affliction is common in persons of the middle of life, who have followed a sedentary pursuit; in women they are more apt to occur during pregnancy, and as a substitute for the menstrual discharge at the period of the climacteric.

Of all forms of varix, none is, probably, of so frequent occurrence as that which affects the superficial veins of the lower extremities, and more particularly the ramifications of the saphena. It is not peculiar to either sex, but is decidedly more common in females than males. This remark does not appear to apply to the continent, however, for we learn from the statistics of Briquet, as well as from the statements of Hasse, that, with them, the male sex is the most liable. Another statement of Briquet's, that it is more frequent in the right than in the left leg, is not confirmed by British experience. Hasse observes that, in men, the dilatation generally arises from the trunk, or the principal branches of the saphena, while he states that, in women, it commences in the minute twigs. It is especially at the ankle, and at the inner side of the popliteal space, that the veins are seen and felt, in the shape of an accumulation of tortuous vessels, of a more or less resistant feel.

A varicose state of the veins of the pia mater is a condition upon which Rokitansky lays some stress, as found after repeated attacks of delirium tremens. Oculists treat of dilatation of the veins of the eye in various forms; and instances of varix in other parts of the upper half

of the body are recorded by authors. Thus, Cruveilhier[1] delineates two cases of varix affecting the arm.

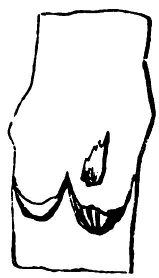
Varicose veins may prove dangerous, by giving rise to hemorrhage, in consequence of ulceration or rupture. They are not, like the arteries, subject to atheromatous disease, though occasionally they become obliterated by the formation of a coagulum, or spontaneous inflammation and cohesion of the parietes.

An obliteration of portions of the venous system, from spontaneous coagulation of the blood during life, is a not unfrequent occurrence, either from the pressure exerted by morbid growths, as aneurisms or cancerous tumors, or without such mechanical causes, from sheer cachectic debility, as, for instance, in a case of empyema that fell under our notice, in which, from the inferior cava downwards, the veins were plugged up with a fawn-colored coagulum. They are not, however, limited to the veins of the inferior half of the body, though most frequently met with in the vena cava inferior, and the portal vein. Dr. Bright[2] records a case in which the longitudinal sinus was filled in a child of twenty months, another of a female aged seventeen, in whom the left jugular and subclavian veins were plugged with a firm coagulum, terminating abruptly just as they entered the cava, and a third, in a female aged twenty, in whom a white, fibrinous coagulum was found in the subclavian vein, extending two or three inches up the jugulars. In both the last cases the hardened veins were traced during life; the subjects were all in an extreme state of exhaustion. In these cases, the coagulum can be easily removed from the channel of the vessel, and the coats of the latter present no evidence of inflammation in the shape of thickening or interstitial deposit, or roughening of the lining membrane. Another form of obliteration is that resulting from a chronic inflammation and consequent adhesive process, set up by the advance of degenerative disease, such as tubercle or cancer. Thus Dr. Lee records two cases of abdominal phlebitis resulting from malignant disease of the uterus; and another of inflammation of the iliac veins in a man, from carcinoma, is related by Mr. Lawrence. Tubercle never directly affects the vessels; it is not found deposited in the coats, nor is it found in their channels. Carcinomatous matter, on the other hand, is very frequently discovered within the veins, either as an immediate extension of the disease, external to them, or as an absorption of the morbid product. Thus, in cancer of the stomach and liver, it has been found in the vena portæ; in renal cancer, in the corresponding vein and the vena cava inferior; in uterine cancer, in the vena cava and its branches. Few of the observers of the cases on record have verified the fact of the cancerous nature of the contents of the vessels by microscopic examination, and in many instances fibrinous coagula have been mistaken for cancer, when coincident with the latter. Langenbeck[3] has, however, established the possibility of the occurrence of cancer within the vessels, by observing cancer-cells in the blood of individuals affected with malignant disease of the uterus. We

[1] Anatomie Pathologique, Livr. xxiii. and Livr. xxx.
[2] Medical Reports, p. ii. pp. 60, 64, 65.
[3] Essai sur l'Anatomie Pathologique. Par. 1816, vol. ii. p. 70.

must not, however, regard the presence of any one variety of cell as essential to determine the malignant character of a morbid growth. The inherent tendency of a certain blastema or matrix is to lead to the production of appearances which are commonly denoted as tubercle, or as cancer, but that blastema in itself need not present any microscopic signs to distinguish it from healthy albuminous or fibrinous deposits. Hence it is not absolutely necessary that we should meet with what are commonly called cancer-cells in the cancerous contents of the veins, in order to justify the conclusion as to their malignant character. Ossification very rarely affects the veins, but cretaceous deposits are occasionally discovered under the lining coat, as in the case of which we have given a delineation. Cruveilhier[1] relates the case of an old man who died of gangrena senilis, in whom the veins accompanying the popliteal artery were studded with phosphatic deposit. This must not, however, be confounded with that variety of concretions termed vein-stones, or phlebolithes, which are met with free in the cavity of the vessels. These are formed of concentric laminæ, of which the internal are hard and brittle, while those forming the outer layers present a softer consistency. They closely resemble the concentric corpuscles so frequently met with in the choroid plexus, where, however, the formation is external to the vein. They are found most frequently in the pelvic veins, and in varices, and appear to result from a stasis in the blood, first giving rise to a coagulum of fibrin, within which a process of cretification takes place; chemically they are found to consist of phosphate and carbonate of lime, bound together by animal matter. The theory of their formation agrees with the mode of explanation which suggests itself for other concentric corpuscles, and is confirmed by what we occasionally see in diseases. Thus, in a case of a large cyst in the kidney, containing, within an inner sac of false membrane, a large black coagulum of blood, we found concentric corpuscles in the false membrane, of exactly the same character as those observed in the brain. Here, too, it seemed reasonable to assume the primary deposition of fibrin, and the secondary precipitation within its laminæ of the phosphate of lime. It is not impossible that phlebolithes may in some instances be the residuary traces of former phlebitis. Dr. Lee observes,[2] that in the spermatic and hypogastric veins of females advanced in life, calcareous concretions and disorganizations of various kinds have frequently been observed, which must have been the consequence of attacks of acute inflammation at remote periods.

Fig. 172.



Calcareous deposit in the coats of a vein.—St. George's Museum, F. a. 12.

ENTOZOA.

Before quitting the pathology of the veins, we have to allude to the presence of entozoa, and of gaseous contents within them. Of the

[1] Medico-Chirurgical Transactions, vol. xvi. p. 418. [2] *Ibid.*

former, instances are recorded by various of the older authors, from Pliny the elder downwards. This writer states, in his *Historia Naturalis*, that animals form in the blood of man, and destroy his body. The most recent observation of parasitic animals in the blood is recorded by Dr. Bushman;[1] but it is liable to objections which tend to invalidate the conclusions arrived at. The observation of the presence of distoma hepaticum in the trunk of the vena portæ in a man aged forty-nine, by Duval,[2] is more valuable and trustworthy. Andral[3] recounts the only instance known of hydatids found in the venous system. They occurred in the pulmonary veins of a man aged fifty-five, and twenty-three occupied the small ramifications shortly before their transition into the capillary network. They varied in size from a pea to a nut, and were symmetrically distributed through both lungs. They had all the characters of acephalocysts.

AIR IN THE VEINS.

The entrance of air into the veins is one of the most formidable occurrences complicating operations about the neck, that the surgeon has to deal with. Death ensues rapidly, and atmospheric air is found in the right side of the heart. Air has been traced in some of these cases in the aorta, the crural arteries, the arteries of the brain, the inferior cava, the iliac veins, and the coronary veins of the heart.[4] It has been suggested that, in some cases of sudden death after delivery, the cause might be found in an introduction of air into the circulation by the open mouths of the veins, when the uterus contracted imperfectly. Another question is the possibility of the spontaneous evolution of gas within the veins during life. Many of the cases on record are undoubtedly mere instances of rapid putrefaction; but we are justified both by the constitution of healthy blood, and by post-mortem observation, in admitting the reality of such a change before death. Numerous authors, among whom we would mention Dr. Baillie,[5] have met with air in the veins of the pia mater in cases of apoplexy, before any traces of decomposition were to be perceived. Dr. Bright[6] attributes the presence of air in these cases exclusively to accidental injury of the veins, or to incipient putrefaction. The evolution of gas during life, though difficult of absolute proof, is entirely within the range of probability, when we consider that venous blood contains an excess of carbonic acid gas, which is discharged on reducing the atmospheric pressure, as demonstrated by Magnus. "Perhaps," as Professor Puchelt remarks, "it happens more frequently than we are aware, that a bubble of air forms in the venous blood, and again disappears. I am acquainted with at least one variety of palpitation, which produces the sensation; and, I am almost inclined

[1] The History of a Case in which Animals were found in Blood drawn from the Veins of a Boy, London, 1833.
[2] Gazette Médicale de Paris, 1842, No. 49.
[3] Magendie, Journal de Physiologie, vol. iii. 69.
[4] See Puchelt, das Venensystem, Liefing, 1843, vol. iii. p. 328.
[5] Morbid Anatomy, p. 430.
[6] Medical Reports, vol. ii. p. 668.

to assert, the noise, as if a bubble passed through a fluid. It occurs generally with but one beat of the heart, and I have met with it in venous subjects with an hemorrhoidal tendency, and a liability to flatulency."

THE CAPILLARIES.

The importance of the capillary circulation in its bearings upon disease, and the relation borne by the blood to the coat of the vessels, and by both to the nervous terminations and other surrounding tissues, can scarcely be over-estimated; and yet we are inclined to think that in most of the experiments performed with a view to determining their functions or ascertaining the part they bear in disease, as in inflammation, the most important of all morbid processes, the share taken by the vessels has been regarded more than the changes occurring in the circulating medium itself. A great physical difficulty presents itself in the examination of the ultimate radicles of the vascular system in most of the organs of the body, from our inability sufficiently to isolate them. Where we are able to do so, as in the brain or pia mater, we may, in inflammation, trace the evolution of the morbid product in the shape of minute molecular spherules, but the coats of the vessels must be looked upon rather as the passive agents of percolation, than as the active promoters of the diseased action. Calcareous deposits are also seen, as in cases of cerebral apoplexy, upon very minute vessels, but we do not possess any means of determining in how far the vascular coats are liable to anything like an idiopathic morbid condition. We need not enter into the question of the part played by the capillaries in nutrition and inflammation, as that is discussed in the general pathology. There are few morbid processes in which they are not involved, though at the same time we must not forget that they are not essential to diseased action, and that, as many healthy changes of the body are affected by metamorphoses directly from the blood, or through the intervention of non-vascular tissue, so, in disease, the capillary system is one of several of the agents of the morbific influence. We must here allude to a form of inflammation which, from presenting somewhat peculiar characters, has been termed by Cruveilhier, capillary phlebitis. It depends upon the same causes as those to which we have traced metastatic abscesses, or purulent deposits, viz: a poisonous infection of the blood, and hence is most commonly met with in organs to which there is a great afflux of blood, as in the lungs, the spleen, the kidneys, and the liver. The form in which it appears, is that of a circumscribed patch of injected and inflamed tissue, which, by itself, may pass through the various stages of inflammation; or it may, in its turn, excite active inflammation in the surrounding parts, which will present different features, in color and consistency, by which the two may be distinguished. Rokitansky observes that capillary phlebitis is not essentially a true inflammation, but that it consists in a coagulation of the blood in some portion of the capillary system, and is analogous to the phlebitis caused by coagulation. He states that the coagulum at first appears as a dark red infarctus of the affected parenchyma; that it subsequently may undergo

various processes, either breaking up and commingling with the blood, or undergoing a retrograde process, leading to obliteration and atrophy of the part affected, or passing into purulent or gangrenous fusion. Rokitansky also expresses it as his opinion that the capillaries may be affected in a similar way as he describes the arteries to be, by an excessive deposition of "lining membrane," only that the anomaly is here less in degree, owing to the arterial portion of the blood being expended in the process of nutrition.

Among the chronic forms of disease which are attributed more particularly to the capillaries, and to which we have not had occasion to allude elsewhere, is the affection which Mr. John Bell, and English writers following him, have termed aneurism by anastomosis, or the Germans, more classically, telcangiectasis.[1] It is also known by the simple term, erectile tumor, which is probably the best, as it implies no theory. The affection is commonly congenital, and presents itself in the shape of a cutaneous swelling of a circumscribed form and bluish-red color, liable to considerable variations of distension, according to the state of the circulation. The tumor commonly, though not always, offers a pulsation to the touch isochronous with the arterial pulse. Bell described the tumor as consisting of a congeries of vessels, between which were cavities and cells communicating with the latter; others have attributed the affection solely to a distended condition of the vessels, among whom may be mentioned Syme and Pelletan; it is, however, extremely probable that both conditions may occur, as in the analogous case of hemorrhoidal tumors; and that while one erectile tumor contains only vessels, another consists of both vessels and cellular cavities. This explanation would aid in understanding the difference in the symptoms presented by this species of tumor.

[1] Teleangiectasis—literally, expansion of the remote vessels; τῆλε, distant, remote; ἄγγος, a vessel; ἐκτείνω, I distend. See also page 175.

CHAPTER XXV.

THE LYMPHATIC SYSTEM.

OUR knowledge of the diseases of the lymphatic system is not commensurate with the importance we attribute to it in the animal economy on physiological grounds. We may infer, from the close relation which it bears to the metamorphoses of the tissues, that it must be morbidly affected in all diseased conditions of individual parts, while its anatomical bearings assist in accounting for the difference which prevails between diseases specially affecting the lymphatic vessels and the veins. A morbid product, or a poison that has found its way into a vein, meets with no impediment, and, unless adhesive inflammation be set up at the point of introduction, speedily taints the whole system; the force and direction of the blood-current materially facilitate the propulsion of any foreign matter that has entered the vein. In the lymphatic vessel we have no such powerful and continuous stream, nor do the channels enlarge in the same uniform manner as in the former; on the contrary, we find the passage every now and then blocked up by a sluice, in the shape of a lymphatic gland, the obvious object of which is to submit the contents of the afferent vessel to a process of purification. By this means, further security is provided against the ultimate introduction into the blood, by the thoracic duct, of deleterious matter, which may have penetrated the lymphatics. Hasse[1] observes that the lymphatic vessels, being exclusively devoted to the purposes of absorption, can contain fluids of very various, and even morbid admixture, without detriment to their internal membrane, and that it is not until the fluids in question have reached the lymphatic glands that inflammatory reaction becomes established. He instances the conveyance of miasmatic and contagious matter through the lymphatic system, as in typhus, the plague, &c., where the lymphatic vessels never exhibit any morbid alteration, although the glands are found more or less disorganized. We must, however, demur both to the fact and to the conclusions drawn by Hasse; for although undoubtedly the mere presence of noxious matters, either in the blood or in the lymph, does not necessitate inflammatory reaction in the coats of the vessels, the fact of lymphatics presenting all the symptoms of inflammation between the seat of irritation and the next chain of glands is one of too frequent occurrence to establish it as a rule that the morbid manifestation only takes place in the latter. The metamorphosing and eliminating power of the lymphatic glands is one that exerts

[1] An Anatomical Description, &c., Sydenham Society's Ed. p. 2.

a most important influence upon the preservation of health, and, where it is weakened, as in scrofulous individuals, we see that every species of disease makes an easy ingress, and is with difficulty expelled. This circumstance offers a satisfactory explanation for the great variation in the susceptibility of different individuals to the action of morbid agents; as we see in the undoubted exposure of several subjects to the same infectant; when, for instance, the same venereal female receives a succession of visitors, we have good grounds for assuming that each of the men came into contact with the syphilitic poison, we may find one enjoying an entire immunity from evil effects, while the other becomes the subject of secondary and tertiary symptoms.

Inflammation of a lymphatic is manifested by redness, painfulness, and swelling, in its course; the coats become thickened and infiltrated, and exudation and suppuration may occur in their channels. A resolution is the most common termination of the process. The presence of pus in the lymphatics does not necessarily demonstrate the existence of inflammation of the vessels; it may be introduced into them by abrasion or ulceration of lymphatics communicating with an abscess. Suppurative inflammation gives rise to small isolated abscesses along the course of the lymphatic vessels, forming, as it were, stations of the disease, each of which appears to serve as a fresh focus of morbid action. The inflammatory process more or less affects the surrounding cellular tissue from the commencement. With the advance of the inflammation, the lymphatics are blended with it, and suppuration and the formation of abscess involve the entire mass. A chronic inflammation of lymphatic vessels is met with in scrofulous, tubercular, and cancerous disease. Their coats are found indurated and thickened, and their channels are blocked up with the morbid blastema of the heterologous growth. The frequency with which this occurs, appears to be in a ratio to the softened condition of the deposit in the organ from which the affected lymphatics take their origin. Thus, in a case of encephaloid cancer of the stomach, in an aged female, that occurred under our own observation, the plexus of lymphatics occupying the lesser curvature of the organ were gorged to the size of crows' quills with the cancerous matter.[1] In the majority of instances, we meet with no such filling up of those vessels, and the subacute inflammation which coexists in their glands appears to be either the result of irritation propagated to them from the primary seat of the lesion, or of idiopathic disease set up in them as the purifying agents of the blood. Sir Astley Cooper[2] reports three cases of obliteration of the thoracic duct, two of which were connected with tubercular, the third with cancerous disease. In the first, the obstruction was produced by the thickened valves, in three distinct parts, adhering to one another, the lowest still allowing of a partial transmission of fluid, the upper arresting it entirely. Scrofulous matter was found deposited between the laminæ of the valves. In the second there was considerable thickening and ulceration of the duct; two fungous growths occupied the channel; and in the third, which

[1] Report of Pathological Society, 1847–48, p. 195.
[2] Medical Records and Researches, 1798, p. 87.

occurred in a man who had died in consequence of malignant disease of the testis and the lumbar glands, the thoracic duct was found much thickened, and filled with a pulpy mass, composed of broken, coagulable lymph. Opposite the curvature of the aorta, the vessel was lost in a swelling as large as a moderate-sized walnut, beyond which it was normal. The paper from which these cases are derived, contains various experiments upon the thoracic duct in animals, of physiological interest. One of the main conclusions arrived at by the author in reference to this point is, that the circulation in the lymphatic system may be kept up by dilatation of collateral vessels subsequent to the occurrence of obstruction in a trunk, as in the vascular system at large.

A varicose condition of lymphatics is occasionally met with in atonic habits, causing them to resemble hydatid tumors. It affects parts of the system, and especially the thoracic duct is liable to dilatation; occasionally the entire system is found in this condition, and an extreme instance of this, occurring in a young man, aged nineteen, which happened in the practice of M. Amussat, is given in the works of Breschet and Carswell. Cruikshank[1] alludes to and delineates a similar case. Here, the lymphatics of the groins had reached a sise sufficient to permit of the introduction of a straw by which air was blown into them; the iliac ganglia had entirely disappeared, and were replaced by the lymphatic vessels. A corresponding enlargement of these vessels was traced through the abdomen into the thorax; none of the other viscera presented any marked pathological changes. The depurating functions of the lymphatic glands while they render these organs safety-valves to the system, also induce in them a frequent liability to disease, which is characteristic of what is familiar to us as the lymphatic constitution. The irritation to which their affections are traceable, may proceed from some local lesions, from which it is carried to the neighboring glands by the connecting lymphatic, as in the case of a sore on the prepuce, inducing bubo, or of porrigo of the head, causing tumefaction of the cervical glands; or, it may be excited directly by the morbid condition of the blood circulating in the capillaries of the glands.

Simple acute inflammation is manifested by tumefaction, softening, and a highly vascularized state of the organ, causing it, on division with the knife, to distil blood, while its color is changed from a reddish gray to a dark red or crimson tint. If suppuration has ensued, yellow spots first appear in different parts, and eventually the entire gland may be destroyed by the process, and be converted into an abscess, in which the surrounding cellular tissue is more or less implicated. The chronic form is, however, the more common; a species of plastic matter is effused interstitially, and induces gradual enlargement and induration of the gland. This condition may be perpetuated, and the resulting hypertrophy exhibits a conversion of the effused matter into the ordinary cell structure, displayed by the microscope as constituting the gland tissue. On the other hand, a reabsorption of the interstitial deposit may occur, as we have frequent opportunities of observing, as the effect

[1] See Carswell, Patholog. Anat. Fasc. ix. pl. iv. fig. 4; and Breschet, Le Système Lymphatique, 1836, p. 260.

of a suitable dietetic and medicinal regimen, in consequence of which the parts regain their normal size and appearance.

Hypertrophy of the glands, whether simple or complicated, with the tubercular diathesis, is peculiarly prevalent in childhood, at the time when the vegetative development of the animal economy makes the greatest claim upon the organs of nutrition and metamorphosis; an atrophic condition is met with in advanced age, after the period of involution has commenced, and it is stated by Rokitansky particularly to affect the mesenteric glands as a result of typhous infiltration, in consequence of which the parenchyma of the gland is absorbed, as well as the product of the process. The inflammation of the mesenteric glands accompanying typhous fever, is a point upon which Rokitansky[1] lays great stress. He considers it as an integral part of ileotyphus, and states it particularly to attack the chain of lymphatic glands corresponding to the affected part of the intestine. He looks upon it as a substantive affection of the glands, allied to the morbid condition which they present in the Oriental plague. The reader will see that, in regard to the lesions of the mesenteric glands in fever, there is a wide difference of opinion between Rokitansky and Drs. Stewart and Jenner, who on very satisfactory evidence have shown that typhus and typhoid fevers are distinct forms of fever, one of the characteristic features of the latter being the intestinal ulcerations and disorganization of the mesenteric glands, while no such affections are met with in typhus.[2]

TUBERCLE.

Few parts of the body are more exposed to the deposit of tuberculous matter than the lymphatic glands. It occurs in them either in a primary or secondary form, as the result of direct elimination from the bloodvessels, or owing to the conveyance of tubercular matter from the organ from which the lymphatics are derived. We see it in the shape of yellowish masses, interspersed among the gland tissue; and as the morbid deposit increases, encroaching more upon, and ultimately entirely destroying all traces of, the normal tissue. The tubercular matter is observed to go through the same processes of softening and suppuration, or of induration and cretification, that we find it liable to elsewhere. Children are peculiarly prone to tubercular disease of the glandular system; but there is a considerable difference in the proclivity of different sets of glands to be affected. All authors are agreed that the bronchial are pre-eminently endowed with this tendency. The analysis of one hundred post-mortem examinations of tuberculous children, by Dr. Lombard,[3] showed that

the bronchial glands	were	affected	in 87	cases.
the mesenteric	"	"	31	"
the cervical	"	"	7	"
the inguinal	"	"	3	"

[1] Pathological Anatomy, Sydenham Society's Edition, vol. iv. p. 390.

[2] For the further details we must refer to the original papers of these authors, which are contained in the Medical Times (1851) and the Edinburgh Medical and Surgical Journal (1840).

[3] Andral, Précis d'Anatomie Pathologique, vol. i. p. 425.

In the case of the bronchial glands, a communication is occasionally established between their contents after they have softened and the channel of the bronchi, and may be thus evacuated, by expectoration. The chalky concretions find their way out occasionally in the same manner. A remarkable instance of death being caused by the impaction of the cretaceous contents of a bronchial gland in the bronchi, is detailed by Dr. Tice, in the twenty-sixth volume of the *Medico-Chirurgical Transactions.*

CARCINOMA.

The lymphatic glands are very commonly the seat of malignant disease; it affects them either primarily or secondarily, but more frequently in the latter form. It is probably owing to the implication of the lymphatic system that we may, in part, at least, attribute the symptoms of cancerous cachexia in an advanced state, as influencing the process of nutrition and assimilation. No other part of the economy is so liable to secondary cancerous deposits as the lymphatic glands; the immediate cause of their becoming the seat of the heterologous growth being the introduction of cancer blastema from the affected organ, or an irritation set up by the proximity of the gland to the latter, which, in its turn, excites a fresh separation of cancer matter from the blood. The second explanation is probably that which obtains in most cases; for we find the parenchyma of the gland to be the prevailing seat of the disease, both in primary and secondary carcinoma of the lymphatic glands. It commonly appears in the shape of a general infiltration, and rarely in isolated nodules or islets. The encephaloid or medullary variety is the form which carcinoma generally presents in the glands. In a case of pancreatic sarcoma affecting the thoracic muscles of the right side, which fell under our own observation, there was a development of a similar morbid growth in the anterior mediastinum, evidently involving, if not proceeding from, the bronchial glands. The tissue was made up of fibres and fibroid cells and circular corpuscles, containing oil-particles. The lungs and other viscera presented no trace of a similar disorganization. In another instance, occurring at St. Mary's Hospital, in which the bronchial glands were converted into a mass closely resembling a malignant growth, though the microscope only exhibited granular corpuscles, resembling the forms seen in tubercle, the lungs were free from substantive disease, though the spleen was both disorganized and hypertrophied, and presented in its tissue deposits resembling those found in the bronchial growth.

MELANOSIS.

An affection to which the glandular system, and especially those parts in relation to the respiratory organs, is very prone, is melanosis; it is scarcely, however, to be looked upon as a morbid process in itself, but rather as an evidence of the depurating functions by which they assist in eliminating the superfluous carbon from the blood. We have shown

elsewhere that melanosis does not, in itself, constitute a malignant disease, and that it does not consist of a new formation, but that it is mainly a secretion of normal constituents of the blood, though frequently complicated with malignant affections. While it is unusual to find black matter in the lymphatic ganglia of the abdominal or inguinal regions, we constantly meet with it in the glands surrounding the bronchi.

ENTOZOA.

The only instance on record, of entozoa being found in the glands, is recorded by Rudolphi.[1] It was found by Treutler, in a person worn out by syphilis; it was an inch in length, tawny in color, semitransparent at one end, presenting two hooklets at its anterior extremity, and hence termed hamularia bronchialis.

[1] Entozoorum Historia Naturalis, vol. ii. p. 82.

THE PATHOLOGICAL ANATOMY OF THE ORGANS OF RESPIRATION.

CHAPTER XXVI.

GENERAL OBSERVATIONS.

We learn from the registers of mortality that the most prevalent cause of death at various ages is to be found, in our climate at least, in morbid conditions of the organs of respiration; if it were possible to estimate the amount of disease introduced into the system through the lungs, though manifested in other organs, we should enhance the importance of an intimate acquaintance with their pathology still further. While the lungs serve the purpose of affording to the blood the means of effecting the changes of assimilation and metamorphosis, this very function renders them, by their extent of surface, and by their intimate relation to the capillary circulation throughout the body, more than any other organ the portal of disease. The impalpable poisons borne on the air enter the human economy chiefly by this entrance, and the system, debilitated by the various influences that wear out its strength, is attacked at this point by irritants of all kinds, introduced directly from without, palpable and impalpable. Thus, the lungs and their accessory parts are subject in various ways to suffer primarily or secondarily from causes that scarcely affect other organs; no age is exempt; no sex or rank offers any immunity; the common air is the common danger, and the sanitary measures necessary to anticipate or remove it, confer immeasurable benefits upon all, though the boon may be but scantily acknowledged.

We shall examine the pathological conditions of the different parts of the respiratory system in the order in which they naturally present themselves, as we proceed from the orifice downwards; commencing with the larynx and its appendix the epiglottis, we shall descend to the trachea, the bronchi and their ramifications, the pulmonary parenchyma, and the pleura. Though a definite relation exists between these different parts, which is more intimate in some than in others, they are each susceptible of isolated morbid states; and though the continuity of the mucous membrane lining the entire passages frequently induces a propagation of disease from one part to the other, this is by no means uni-

versally the case, and the catarrh or croup affecting the larynx or trachea need not cause any pathological changes in the adjacent mucous membrane, or the other tissues of the affected part itself. The absolute importance of the morbid condition to the individual, does not necessarily bear a direct relation to its intensity, but depends a great deal upon the part affected; a point which the physiological laws regulating the different sections of the respiratory organs render sufficiently palpable; thus, a trifling amount of inflammatory swelling of the glottis or epiglottis threatens danger, while much more intense inflammation of the pulmonary parenchyma, or of the bronchi, may run its course without causing more than a temporary inconvenience. The features of the morbid condition may be identical, while their bearings upon the existence of the individual are widely dissimilar.

THE EPIGLOTTIS.

The epiglottis is, notwithstanding its exposure, not very liable to disease, a circumstance due in part to the dense fibro-cartilaginous tissue which forms its substratum. The mucous membrane which invests it, may be affected with acute or chronic inflammation, in which case the vessels enlarge and become tortuous and congested, and the light pink hue is converted into a streaky, or more or less uniform redness. The

Fig. 173.

Acute ulceration of the epiglottis and surrounding parts in a man, caused by taking a large dose of bichloride of mercury. Death followed after nine days; there were pneumonia, ulceration of the stomach, and inflammation of the entire intestinal tract.

acute form is commonly the result of mechanical injury or chemical irritation; while the chronic form accompanies old standing catarrhs of advanced age, arthritic or syphilitic cachexia, and other conditions resulting from general atony. The former is met with as a result of the action of irritant poisons, which may thus cause death without pass-

ing the fauces; and as Dr. Marshall Hall[1] has pointed out, in consequence of children, as frequently happens among the poorer orders, drinking boiling water from the spout of the kettle or tea-pot. In these cases there is also rapid and extensive œdema, giving rise to a mechanical impediment to the admission of air into the lungs. In one of the instances detailed by Dr. Davis,[2] suffocative dyspnœa supervened within three or four hours, and when at this period the attempt was made to apply leeches, the child, a little girl of three years of age, was much terrified, and screamed so violently, that they could not be applied. From this moment, however, respiration became easy, and a speedy recovery took place, owing most probably to a rupture of the bullæ which had formed in consequence of the irritation. The remedy which in such a case relieves the immediate symptoms, and may thus be the means of saving life, is the operation of tracheotomy, though, as in the instance from which the delineation was taken, not always successful.

Ulcerations of the epiglottis are not unfrequent, more especially as complications of phthisis; they are generally limited to the inferior surface of the valve and to its mucous covering; though they occasionally penetrate to the fibro-cartilage. They are not generally accompanied by much tumefaction or reddening of the surrounding tissue. Louis[3] states that in all the cases that fell under his notice, he only once observed ulcerations on the lingual surface; he found the breadth of the ulcers to vary from about one to two lines; though in some cases the laryngeal surface of the epiglottis was entirely deprived of its mucous membrane: in four cases the edge of the fibro-cartilage was destroyed, as well as the surface ulcerated, so as to give the part a festooned appearance; in a fifth case the epiglottis was totally destroyed. The proportion of phthisical cases in which the epiglottis is affected, appears, according to the same author, to be about one-quarter. These ulcers are not, however, the result of a fusion of tubercular deposit, which is not met with at this point, but of sympathetic irritation and inflammation; they, like other affections of the mucous membranes of the air-passages which have been mistaken for tubercular deposit, often originate in occlusion and sebaceous enlargement of the numerous solitary follicles scattered about the respiratory mucous membrane. These vary in size from an almost imperceptible point to a pin's head, and even to that of a bean;[4] and closely resemble miliary granulations, surrounded by a halo of bloodvessels, as they appear for instance in the pia mater. The microscope, however, reveals their structure; showing the basement membrane of the follicle lined with epithelium, and containing oily matter. The cheesy particles coughed up by many people, are formed partly in the follicular structures of the tonsils; and probably also in the follicles of which we have just spoken. The epiglottis is liable to suffer from syphilitical ulceration, by an extension of the disease from the fauces: it very rarely passes beyond to the larynx and trachea. In smallpox and other eruptive fevers, the peculiar poison of the disease

[1] Medico-Chirurgical Transactions, vol. xii. p. 1.
[2] *Ibid.*
[3] Researches on Phthisis, Sydenh. Soc. Ed. p. 42.
[4] See a case detailed in Dr. Bright's Reports, ii. p. 644.

often sets up its action in the fauces and the respiratory passages; the former may give rise to pustules on the glottis, accompanied by more

Fig. 174.

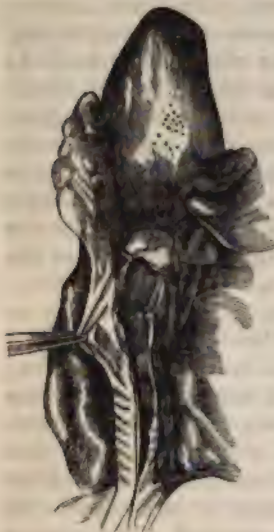


Fig. 175.

Fig. 176.

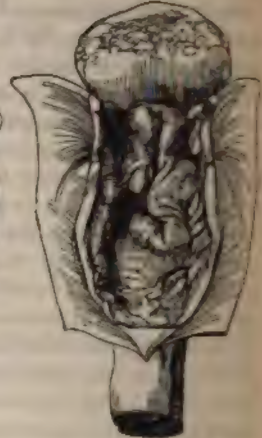


Fig. 174.—Œdema of the epiglottis, brought on by drinking scalding water, and causing the death of the child, a boy aged three years and nine months, in two hours. The uvula, which is seen a little above and to the right of the epiglottis, was also œdematous. No other feature was observed, except some reddening of the bronchial mucous membrane, and congestion of the lungs. Tracheotomy was performed.

Fig. 175.—Œdema Glottidis.

Fig. 176.—Acute Œdema Glottidis; exposed from behind.

or less redness and serous infiltration; in the latter, ulcerative destruction and œdematous swelling occur. It is to an affection of these parts that more particularly the danger of the retrocession of the cutaneous affections is attributable: there is frequently an inverse ratio between the force with which the external and internal symptoms are manifested.

THE LARYNX.

The mucous membrane of the larynx presents the same changes that we meet with in the mucous membrane investing the entire respiratory tract. In health it has an almost colorless, slightly pink hue, and consists of a uniform epithelial surface, overlaying a basement membrane, underneath which the vascular network ramifies. The surface is broken only by the minute follicles which open upon it. In congestive and inflammatory states this vascular network becomes much distended; the membrane is rendered thick and soft by mere repletion in the first instance, and subsequently by interstitial effusion. The more lasting and chronic the inflammatory action, the more this thickened condition is established, and thus a permanent hypertrophy of the membrane is effected. The color of an inflamed respiratory mucous membrane varies

according to the intensity, and also according to the character of the inflammation, from a bright crimson or scarlet, to deep purple or dusky redness. The hyperæmia is often found to be entirely local; this is chiefly the case in chronic affections: thus, we may find it limited to the larynx, the trachea, or the bronchi. The character of the inflammation necessarily determines the nature of the products which occur upon and beneath the membrane. The most acute form is probably that in which, from the contact of a powerful irritant, such as boiling water, an instant effusion of serum takes place in the submucous tissue, causing its distension or œdematous swelling. We have already alluded to the cases in which children have met with their death in consequence of drinking the hot contents of a kettle from its spout. In some of these it appears that the œdema rather affects the parts below the epiglottis than the epiglottis itself. Here, it is rather the mechanical effect of the tumefaction of the loose tissue at the glottis than the intensity of the pathological process, that produces the fatal issue. In catarrhal inflammation of the larynx, the dyspnœa, and affection of the voice, depend mainly upon the amount of tumefaction of the rima glottidis; during the first stage there is, as elsewhere, in the mucous membrane a sense of titillation, roughness, and pain; and as the stage of secretion supervenes, these symptoms subside and are relieved by expectoration. This presents various appearances to the naked eye; at first, it is a glairy viscid mucus, which subsequently assumes a greenish or yellowish color, losing its adhesiveness and forming into opaque rounded pellets. If a blennorrhæic state supervenes, the discharge assumes a purulent character. Under the microscope these varieties can scarcely be distinguished. In each, we find mucous corpuscles and epithelium; and if the inflammation has been of sufficient intensity to cause hemorrhage, we shall also discover blood-corpuscles. Idiopathic laryngitis appears generally to be the result of atmospheric changes; it runs a rapid course, and it is one of those affections in which the performance of tracheotomy may become the means of saving the patient's life. The disease acquires an historical interest, from the circumstance that it proved fatal to Washington. Rokitansky describes, under the name of gonorrhœal catarrh of the larynx, a variety which he attributes to metastasis; he considers it important on account of its leading to a contraction of the passage. He states it to attack the mucous membrane of the epiglottis and the duplicatures of the glottis, converting the mucous membrane and subjacent areolar tissue into a fibro-lardaceous white resistant structure of tolerable thickness, thus giving rise to contraction of the rima glottidis and the cavity of the larynx.

In children, catarrhal inflammation of the larynx may produce all the symptoms of croup; in which case, though, as Andral observes, the passage of air through the contracted rima gives rise to the well-known sound of croup, the post-mortem examination will only exhibit a slight tumefaction of the mucous membrane, without any trace of the membranous exudation, which Bretonneau, Copland, and others consider as characteristic of the disease. On this subject, the morbid anatomist still continues at issue with the nosologist, a circumstance which must be attributed to the unwillingness, not to call it by a stronger term, of

the former to recognize any other proof of disease than that discoverable by the scalpel. It appears difficult to avoid seeing that the peculiar disposition existing in infancy to spasmodic affections, dependent upon great irritability of the excito-motor system, may convert the most trifling irritant into a cause of danger and death, which would not be demonstrable by our present means of examination; even in marked cases of diphtheritic exudation, the child does not always die of the mechanical obstacle, but by the indirect influence it exerts in producing glottic spasm. The variations presented by croup are numerous, and depend upon atmospheric causes, or upon peculiarities of living, regimen, and residence.[1]

The larynx is the most frequent seat of croupy exudation, and though occasionally found to extend even into the smaller bronchial ramifications, the deposit in the trachea and bronchi appears invariably to be continuous with the laryngeal exudation. On the continent, more commonly than with us, the exudation also forms on the velum palati, and in the fauces and pharynx; according to Bretonneau's[2] observations during an epidemic at Tours, the exudation occurred simultaneously in the gullet and air-passages, in fifty cases, while in one only the former was unaffected. Six or seven times he found that the membranous concretion extended to the smallest bronchi, and in one-third of the entire number it reached beyond the main division of the bronchi; in all the rest it terminated at different points of the trachea. In this form it has received the name of diphtheritis, or pellicular inflammation. The exudation itself is of an albuminous character, of greater or less consistency—we have seen it of a translucent jelly-like character, scarcely adherent to the parietes—though commonly it presents the appearance of a thin and tolerably firm layer, moulded upon the mucous membrane, varying in thickness from a mere film to a coat a line and a half in diameter. It then exhibits the ordinary appearance of fibrin; of a light yellow or cream color; more or less intimately attached to the mucous membrane; from the latter it may, however, always be separated, leaving its surface entire, and generally of a bright red. The functions of the mucous membrane appear to have undergone an entire revolution; the secretion resembling rather that of a serous membrane in tenacity, adhesiveness, and coagulability. It is deposited in patches, which may gradually coalesce, and thus form rings or channels of greater or less extent; they send off small prolongations into the follicles scattered over the mucous membrane, by which they are secured in their place until a suppurative process underneath, loosens them, and allows them to be

[1] The reader is particularly referred to the nineteenth and twentieth Lectures in Dr. West's work on the Diseases of Infancy, &c.

Croup is a disease known by a great variety of names, which are calculated to embarrass the student. Cynanche is the oldest term, under which it is described by Paulus Ægineta, as a well-known affection (see the Sydenham Society's Edition of his work, vol. i. p. 464); modern writers term it promiscuously cynanche, angina, trachitis, with the epithets membranacea, polyposa, stridula, diphtheritis, pellicular inflammation, asthma acutum infantum, &c. The English name croup is of Scotch origin, and was first, we believe, introduced into general use by Sir Everard Home.

[2] Des Inflammations Spéciales du Tissu Muqueux, &c., par P. Bretonneau. Paris, 1827, p. 32.

thrown off, if the patient retains strength to do so. No chemical or microscopic difference has hitherto been shown to exist between croupy exudation and the product of acute inflammation of serous membranes, or of the fibrin of the blood. The hue of the subjacent tissues in cases accompanied by great disturbance of the circulation, or with typhoid symptoms, becomes dusky and livid. There rarely are any abrasions of the mucous membrane of the trachea. If there is more than usual difficulty in removing the false membrane from the larynx, this depends, as Dr. West[1] remarks, upon the more extensive alterations which this part of the air-tube has undergone. It is generally red and swollen, especially about the edges of the rima glottidis, the arytænoid cartilages, and the openings of the sacculus laryngis. It is in the former that we occasionally find small aphthous ulcerations. In that form of croup which succeeds to measles, we are informed by Dr. West, that there is, in many instances, a condition of unhealthy inflammation, and aphthous ulceration of the mouth and gums; a slight speck of ash-colored false membrane covering each little ulcer.

The diseases with which croupy inflammation is most frequently complicated are bronchitis and pneumonia; though we very commonly meet with no other symptoms of morbid action than those found in the part affected; nor is there any doubt that these may be so slight as altogether to escape observation, in the post-mortem examination. We must here, as elsewhere, be careful not to mistake the pulmonic congestion, resulting from the suffocative influence of the malady, with inflammatory action.

The disease which we have just considered is almost exclusively a disease of early childhood. The aphthous ulcers found as a result of aphthous stomatitis, or muguet, are equally peculiar to infancy. It is not so with the ulcerations which constitute an affection termed phthisis laryngea, which accompany tubercular phthisis of the lungs, and rather belong to puberty and the later periods of life. It has been long thought to constitute an idiopathic disease, but morbid anatomists are now agreed that it occurs only as a sequela of tubercular disease of the lungs, and also that it is very questionable whether it is ever preceded by the deposit of tubercle in the larynx itself. With regard to the latter point we must not, however, overlook the statement of Rokitansky, that it is deposited in the form of gray granulations in the submucous areolar tissue, or as yellow caseous matter, infiltrated into the mucous membrane. He has found it constantly and exclusively in the vicinity of the transverse muscles and the adjacent arytænoid cartilages. His experience is strikingly at variance with that of Louis, who has not in a single instance met with tuberculous granulations in the substance, or on the surface of the epiglottis, larynx, or trachea. Dr. Watson attributes these ulcers exclusively to the acridity of the sputa; but, although the sputa manifestly exercise an influence in their production, there must be some further cause, as they are by no means constant accompaniments of phthisis, and as they do not form at some points, *e. g.* the ventricles of the larynx, in which a lodgement of sputa must constantly take place. Louis states ulcers in the larynx to have occurred in a fourth part of his cases.

[1] Lectures on the Diseases of Infancy, 2d ed. p. 221.

In children, ulceration of the air-passages is scarcely ever met with; Dr. West[1] has only seen it once in early life; on that occasion, several small excavated erosions were found, just above the chordæ vocales, in a child of twenty months, besides a general redness of the bronchial tubes. In the larynx, their site is generally at the junction of the vocal chords, on the vocal chords themselves, and on the arytenoid cartilages.

Ulceration of the mucous membrane may extend to the cartilages of the larynx; or these may be primarily the seat of inflammatory action, as the result of deep-seated cachexia, such as syphilis. To this cause we must also refer the epithelial formations or polypoid growths, which are occasionally found springing from the mucous lining of the larynx. Cancer nodules of the medullary variety are also met with in these parts, and Rokitansky describes, as a very singular form, a cancerous degeneration of the arytenoid cartilages. These affections are generally accompanied by symptoms of chronic laryngitis, consisting in reddening and serous infiltration and hypertrophy of the surrounding tissues; with wasting and degeneration of the laryngeal muscles, and occasionally the formation of abscesses in the submucous layer.

Fig. 177.

Ulceration of the larynx.

The laryngeal cartilages have a tendency to ossify in advanced life in obedience to the general law affecting similar structures; but this metamorphosis is also produced by disease; and it is probably in those cases of chronic laryngitis chiefly, which are connected with a rheumatic diathesis and originate in the perichondrium, that this metamorphosis takes place. The larynx is not so liable to be affected by tumors or foreign bodies pressing upon it as other parts of the respiratory system, on account of the greater rigidity of its walls, and the capaciousness of its interior. Foreign bodies arrested in the œsophagus generally become impacted behind the larynx; when introduced by the glottis, they may become fixed in the chink; but if they pass the portal they necessarily drop through the wider cavity of the larynx, and become fixed at some lower point, or play up and down in the trachea.

THE TRACHEA.

Many of the observations which we have had occasion to make on the pathology of the larynx apply to the trachea. The congestion of the mucous membrane presents similar characters; it is generally observed

[1] The Diseases of Infancy, 1st ed. p. 71.

to be most marked in the membranous interstices, between the rings and at the posterior part. Occasionally, it is bounded by a sharp outline; thus, where the hyperæmia of the trachea is associated with disease

Fig. 178.

Enlargement of follicles of mucous membrane of the trachea, closely resembling miliary granulations, and accompanying pulmonary phthisis. The drawing on the right is an enlarged view of a single follicle, surrounded by enlarged and congested vessels. The glandular structure was distinctly apparent when examined under a higher power.

of one lung, it may be seen on the corresponding half of the former only, the mesian line forming the division between the normal pale tissue and the portion that presents increased vascularity. A similar observation has been made with regard to the occurrence of ulcers, viz: that when following unilateral pulmonary disease, they are confined to the corresponding side of the trachea. Otherwise, the point of divergence of the bronchi and the posterior mesian line are the most frequent seat of tracheal ulcers. They may often be traced to follicular inflammation, in the same way as obstructed solitary follicles put on the appearance of tubercular deposit. The essential connection between ulceration of the trachea and pulmonary phthisis is even more firmly established than in regard to the relation between the latter and laryngeal ulceration. Louis[1] found that, of 190 phthisical subjects, seventy-six presented ulcerations in the trachea; while in 500 non-phthisical subjects, carried off by chronic diseases, examined by the same pathologist, not one presented any tracheal or laryngeal ulceration. A curious circumstance connected with this question is the different ratio in which the two sexes are affected; thus, Louis has shown that, while only about one quarter of the females who succumb to pulmonary consumption exhibit ulcers in the trachea, they are found in half the male subjects similarly diseased.

Croupy inflammation of the trachea presents no characters which differ from the phenomena exhibited by the disease when affecting the larynx alone.

The rings of the trachea, like the cartilaginous structures of the

[1] On Phthisis, Syd. Soc. Ed. p. 42.

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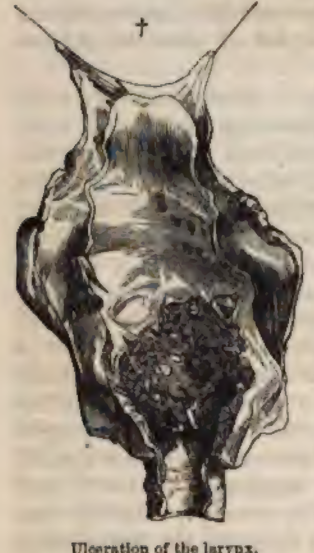

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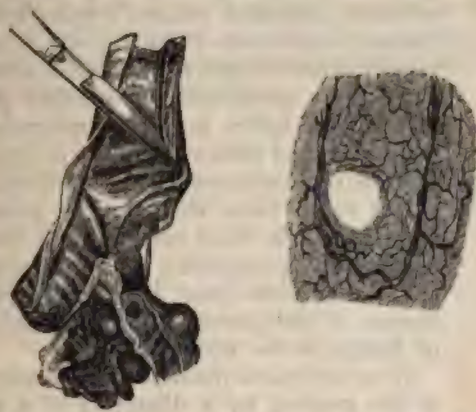

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larynx, are liable to become abnormally ossified, a condition which does not in itself exert any material influence on the health of the individual. The functions of the trachea are more liable to be interfered with than those of the upper portion of the air-tubes, by pressure exerted from without; encysted tumors, hypertrophy, and other diseased conditions of the thyroid gland, aneurismal tumors of the carotid or subclavian arteries, or of the arch of the aorta, enlarged cervical glands, tumors, and foreign bodies in the œsophagus, phlegmonous erysipelas and diffuse abscesses in the vicinity, may each induce compression of the windpipe to such an extent as to cause death. The practical question which arises in these cases regards the propriety of performing laryngotomy or tracheotomy; it is also one that requires great judgment in connection with disease of the mucous membrane, and the presence of foreign bodies within the tube.

Fig. 179.

Example of false membrane in croup.

Diverticula are described as occasionally forming in the trachea, by a dilatation of a portion of the posterior wall, and consequent protrusion into the œsophagus between the ends of the tracheal rings; they have been attributed, by Rokitansky, to an hypertrophy of the muciparous glands. The dilated duct of the hypertrophied cysts may be discovered in the pouch; which confirms the explanation of its origin. When met with in the trachea, a similar condition is sometimes traceable in the bronchial tubes.

The presence of extraneous matters in the air-passages is always an occurrence of very serious import; but, unless the substance becomes impacted in the rima glottidis, it is not immediately fatal; the mucous membrane of the lower portions being less irritable and less liable to produce spasmodic action. The more rough the surface of the foreign body, the sooner inflammation is likely to supervene; and, unless it be removed by coughing, or by an operative proceeding, the issue is certain death. If of a globular form, and too large to enter into the bronchi, it will pass up and down in the trachea, giving rise to a variety of sounds. Cherry-stones, buttons, coins, teeth, morsels of food, portions of bone, and other things, have thus found their way into the trachea, and remained there or got impacted in the bronchi. One case is recorded by Mr. Liston,[1] as a solitary instance, of a piece of bone having lodged in the right bronchus (which, from its size and relation to the trachea, generally becomes the receptacle for foreign bodies), and having been discovered and removed during life. A triangular piece of mutton-bone had, six months previous to the operation, become entangled in the glottis, and shortly after slipped down the trachea. A perfect recovery resulted.

The upper portion of the air-tube is also liable to fatal injuries inflicted accidentally or intentionally. In those cases in which life is not imme-

[1] Practical Surgery, 3d ed. p. 412.

diately destroyed by hemorrhage, the injury may prove fatal by the secondary inflammation and tumefaction of the mucous membrane, or by coagula obstructing the passage of air. The latter is particularly likely to happen as a consequence of hasty surgical interference, in prematurely sewing up the wound, and thus preventing the necessary escape of the blood. The trachea is less frequently subject to become the seat of condylomatous and epithelial growths than the larynx; the same applies to cancer, though carcinomatous affections of the neck occasionally make their way into the trachea.

CHAPTER XXVII.

THE BRONCHIAL TUBES.

THE diseases of the bronchial tubes affect the individual very differently, according as the larger or smaller divisions are the seat of the morbid action. Thus, the same amount of tumefaction which, in the mucous membrane of the bronchi near the trachea, will scarcely give rise to any inconvenience, will, in the finer ramifications, be the cause of intense dyspnœa and danger. The terminal points of the respiratory system, in this respect, resemble each other; an acute tumefaction about the aperture of the larynx, and at the opposite end of the bronchial tree, may equally induce suffocative symptoms. Moreover, it is not always easy to determine the limits of bronchial and parenchymatous disease of the lungs; and it is scarcely possible that inflammatory affections of the latter can take place without involving the smaller bronchi. Hyperæmia of the bronchial mucous membrane is a phenomenon of every-day occurrence, accompanying catarrh, gastro-intestinal, hepatic or cardiac affections, and manifested in its most marked forms by hemorrhage. Hæmoptysis is rarely idiopathic as a mere result of plethora, but commonly associated with a deeper-seated morbid affection; a crasis subsequently manifesting itself by further disorganization, or an organic disease, which has already been discovered. Pathologists have not yet succeeded in demonstrating the exact manner in which capillary hemorrhage takes place; in a few instances of advanced tuberculosis, patulous vessels which were eroded by the progress of the ulceration, have been discovered, to which pulmonary hemorrhage could be attributed; but the common process by which the blood is discharged, is undoubtedly analogous to the "sweating" of blood by which the menstrual flow has been observed to be effected; it is rather by exhalation or secretion that the overloaded vessels relieve themselves, than, as the term hemorrhage[1] implies, by laceration or rupture. The hemorrhage is not a mere passive occurrence, resulting from a retardation of the vascular current, or we should find it accompanying hypostatic congestion, which it does not; it must be looked upon as an evidence of further disease of an active character, as a symptom calling for our careful attention, but only to be treated in reference to a fundamental affection. Hemorrhage from the lungs by exhalation rarely proves fatal in itself, though at times the amount of blood lost is very considerable. Dr. Copland quotes the case of a patient who lost about 192 ounces in twenty-four hours, and reco-

[1] Hemorrhage, etymol. αἷμα, blood; ῥήγνυμι, I rupture; ῥαγὰς, a cleft, a fissure.

vered. The frequency with which it is associated with phthisis, has been determined by Louis to be about two-thirds of all the cases; a relation which is confirmed by recent observers, and one which, taken with other circumstances, casts much suspicion on the nature of the cases of so-called vicarious hemorrhages, whether they take the place of the menstrual or hemorrhoidal discharge. With regard to the former, we cannot overlook the very frequent derangement of the ovarian function in company with tubercular disease of the lungs; and if we add to this the demonstrated connection between hæmoptysis and phthisis, it is difficult to avoid seeing that vicarious menstruation of this character is almost necessarily indicative of a tubercular tendency. The difficulty of explaining the mode in which hæmoptysis takes place, is as great in regard to the stages of softening as in the earlier stage of crudity; the vessels become obliterated in the former instance as the tubercular matter and pulmonary tissues deliquesce, and we are rarely able to discover the open mouths of lacerated vessels. At a future page, we shall also have occasion to see that the hemorrhage into the tissue of the organs constituting pulmonary apoplexy, is distinct from that bronchial hemorrhage with which hæmoptysis is commonly associated. In either stages, therefore, of tubercular disease, it remains for us only to assume that the congestion takes place on the bronchial mucous membrane, and that the discharge which ensues, is a symptom of the blood crasis rather than of the local affection to which it has given rise. The relation of hæmoptysis to the different stages of phthisis, is one to which Dr. Walshe[1] has especially directed his attention; the results of an analysis of the cases which had occurred at the Hospital for Consumption at Brompton, in reference to this question, are as follows:—

	Number of cases.	Frequency of Hæmoptysis.	
		Absolute.	Per cent.
First stage	39	28	71.79
Second stage	20	18	90.00
Third stage	69	57	82.61

The proportion changes somewhat, when the analysis is made for each of the sexes; we then find that the increase of hæmoptysis during the second and third stages is considerably greater in men than in women, which may fairly be explained by the greater bodily labor, and increased tax upon the pulmonary circulation, in the former than in the latter; it rather tends to show that the exciting influence of the tubercular cachexia itself, in producing the hemorrhage, is greater than that of its secondary results. The following is the table illustrating this point:—

[1] Medico-Chirurgical Review, vol. iii. p. 225.

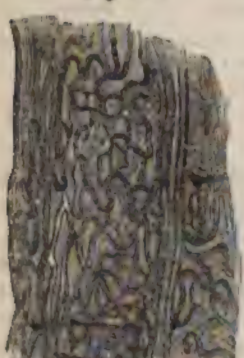
	MALES.	HÆMOPTYSIS.		FEMALES.	HÆMOPTYSIS.	
	Number of cases.	Absolute frequency.	Per cent. frequency.	Number of cases.	Absolute frequency.	Per cent. frequency.
First stage	18	12	66.66	21	16	76.19
2d and 3d stages	56	49	87.50	33	26	78.78

We forbear entering into a further disquisition of this point, as it would carry us beyond our prescribed limits; we cannot, however, avoid pointing out, that it bears most immediately upon the question of the treatment of the early stages of phthisis, for if, as we would conclude, the hemorrhage is a local symptom of the general cachexia, the indications would be to remove the local congestion by non-debilitating counter-irritation, while we correct the constitutional taint by the remedies which we know to be capable of doing so. Neither, alone, would be likely to suffice, nor would anything appear more objectionable than the employment of remedies applicable to a state of plethora and general vascular excitement; we allude more particularly to venesection and mercurials.

BRONCHITIS.

In inflammation of the bronchi we find the same variations of injection and secretion that are presented to us in similar conditions of the upper portions of the air-conduit; but the nearer we approach the terminations of the subdivisions, the more the bronchules will be found filled with the fluids poured into them, corresponding in character, in a measure, to the sputa seen before death, but with a greater admixture of air, the less viscid and tenacious the secretion. The post-mortem appearances may be limited to mere redness of the mucous membrane. In the smallest subdivisions, we must be careful to discriminate between the injection of the bronchules, and the redness resulting from the translucency of their tissue, allowing the color of the subjacent pulmonary parenchyma to shine through. The redness is generally tolerably uniform in the part affected, fading off at the margin into the healthy tissue; we do not commonly meet with that arborescent or punctiform injection in the bronchi, which is seen in inflammations of other mucous membranes, as that of the stomach. Sometimes the affection resides exclusively in the larger bronchi, fading off in the smaller divisions; at others it occupies the reverse relation; the danger to the individual increasing with the number of small tubes affected; the tumefaction and loss of elasticity in which, necessarily exert a great influence in

Fig. 180.



Injection and stasis in the vessels of the bronchial mucous membrane, in bronchitis, seen by a low power. The vessels were disposed in longitudinal clusters, united by transverse inosculations.

producing dyspnœa. The actual sense of the difficulty of breathing, as well as the real absence of proper aeration of the blood, shown by the lividity of the patient, has appeared to us to be greater in these cases of capillary bronchitis than in pneumonia. The more asthenic the form of bronchitis, the more the redness of the bronchial mucous membrane approaches a livid purplish tint; it is generally found of this hue in the chronic forms. The secretions will vary according to the stage and character of the disease, from a viscid glazy mucus, to a genuine purulent discharge of a more or less diffluent character. Occasionally, death is the result of a sudden effusion of liquid into the bronchi, constituting what is called suffocative catarrh, which is met with more frequently in the infant than in the adult. Long-continued purulent expectoration may, however, have existed during life, without any appreciable lesion being discovered after death; in these cases, the bronchial mucous membrane, as Andral[1] observes, need not even present a trace of redness. In examining the lungs, we must be careful to compare different portions, before arriving at definite conclusions; for it is often difficult to determine to which part the fluids belong which exude on section. The surest way to ascertain the state of the parietes and contents of the bronchi, is to follow them from the larger trunks with a pair of scissors, carefully avoiding to admit more extraneous matter into the tubes than we can help.

Croupy inflammation may affect the bronchi, as it does the upper respiratory passages, though it does so with less frequency; complete moulds of portions of the bronchial tree present themselves to us; the influence they exert upon respiration depends partly upon the obstruction they themselves offer, and partly upon the tumefaction of the subjacent mucous membrane.

The mucous membrane of the bronchi, like the mucous membranes of the urino-genital organs, occasionally exhibits a chronic affection, in which, without marked symptoms of an inflammatory character, the membrane pours out a plastic exudation, which forms what has been termed bronchial polypi. Their microscopic characters have not as yet been determined, but, if a surmise may be hazarded, we should expect to find them consisting of epithelium, matted together by an unusually viscid mucus. Dr. Watson observes, that though it is surprising that patients should recover from the affection, it never in itself seems to prove fatal. Dr. Reid[2] has reported two cases of tubular expectoration from the bronchi, occurring in the adult, with delineations, which closely resemble that given by Dr. Baillie, in his work on morbid anatomy. In one, the patient, a married lady, aged twenty-eight, affected with a chronic cough, consequent upon an attack of bronchitis, frequently, after suffocative attacks, coughed up arborescent membranous substances, resembling casts of the minute bronchial tubes: the second case occurred in a gentleman, aged forty-four, and closely resembled the former, except that there was more manifest congestion, and that the casts were more of a sanguineous character, and their rejection each

[1] Précis d'Anatomie Pathologique, ii. 481.
[2] Medico-Chirurgical Transactions, vol. xxxvii. p. 333.

time accompanied by some hemorrhage. It appears to be more frequent on the continent of Europe, where the affection has carried off several distinguished individuals, among whom the Empress Josephine is the most eminent.

Other morbid states, besides those already alluded to, may give rise to a constriction or stenosis of the bronchial tubes; it may be produced by an actual hypertrophy of the submucous layers, as a result of chronic bronchial irritation, or by serous effusion, as in dropsical states of the system. The physical symptoms in these different cases may be identical, being produced by analogous structural alterations; but it is manifest that the constitutional basis upon which they rest may differ very considerably, as also the influence they exert in living subjects upon the further production of morbid conditions.

The pulmonic symptoms accompanying typhous and typhoid fevers, is set down by Rokitansky[1] among the class of catarrh of the mucous membrane; he states it always to appear as an intense diffused congestion; the mucous membrane is of a dark, almost violet tint, swollen and succulent, and yields a secretion of a gelatinous and sometimes dark, blood-streaked mucus. The disease, according to this author, is most commonly developed in the bronchial ramifications of the lower lobes; it is always limited to the stage of typhous congestion, and never gives rise to any apparent production of a secondary formation on the tissue of the mucous membrane, such as is produced in immense quantity in the intestinal follicles in cases of abdominal typhus. He based his diagnosis of a primary broncho-typhus, in which the disease is localized here to the exclusion of all other mucous membranes, upon the peculiar stasis and swelling of the spleen, and at the cul-de-sac of the stomach, the remarkable character of the blood, the typhous nature of the general disease, and especially the altered condition of the bronchial glands. The last lesion of the bronchial mucous membrane which remains for our consideration is, ulceration; as in the larynx and trachea it is mainly, if not exclusively, a concomitant of phthisis. It has been observed by few authors, probably on account of the care requisite in the examination, to discover an abrasion in these parts. Louis, who examined the bronchi of forty-nine phthisical subjects, with special regard to this point, found ulcerations in twenty-two. Dr. Copland is of opinion that ulcers occasionally perforate the bronchial tubes, and thus occasion abscesses in the pulmonary parenchyma.

The submucous tissues may be variously affected in the bronchi, as in air-passages already considered. The cartilaginous rings may undergo a process of softening or the opposite condition of ossification; in this case, they become brittle, and break; they then either project like fish-bones, as Andral observes, into the bronchial cavity, or, becoming detached, are expectorated.

[1] Pathological Anatomy, vol. iv. p. 23, Syd. Soc. Ed.

DILATATION OF THE BRONCHI.

Many of the lesions which we have adverted to may co-operate in producing a morbid condition of the bronchi, to which Laennec was the first to draw attention, and which, though of extreme importance in a nosological point of view, has hitherto been rather treated as a question

Fig. 181.

Dilated bronchi, from a female aged 52, who had suffered from chronic pneumonia and bronchitis for three years; the pulmonary tissue intervening between the bronchi was much condensed.

affecting the pathologist than the practitioner. It is dilatation of the bronchi or bronchiectasis.[1] By Laennec, it was attributed exclusively to an accumulation of mucus in the ramifications of the dilated portion; but, as Dr. C. J. B. Williams justly observes, if this were the cause we should not, as we do, hear the air penetrate freely into the dilated portions. There may, undoubtedly, be various efficient causes at play in the production of this diseased state, both of a mechanical and of a more dynamical character. The most palpable instance of the former is presented to us in the case of an enlarged bronchial gland, whether it contains cretaceous or simply scrofulous matter, compressing a bronchus. Here, the free exit of the respired atmosphere being prevented, an accumulation of air might be supposed to take place behind the narrowed portal, the channels being prevented ever collapsing to the same extent as a healthy lung. Any impediment to the entrance or exit of the air into the lungs will produce irregular and forcible breathing, and throw a greater strain upon those parts especially, which are in the vicinity of the obstacle.

One point having yielded, it is quite intelligible that the distension should gradually progress, while, at the same time, it must, in a corresponding ratio, compress and gradually obliterate the surrounding

[1] Etymology—βρόγχια, the bronchi, and ἔκτασις, dilatation, from ἐκτείνω, I stretch. We may take this opportunity of remarking upon the distinction which some authors have lately made between bronchia and bronchi; it is a source of some confusion, and scarcely warranted by their etymology of the words; if a diminution is required, the term bronchule is more convenient, and not liable to be mistaken.

pulmonary tissue. In the majority of instances a diseased condition of the bronchial parietes, if not, as Corrigan has suggested, of the pulmonary parenchyma itself, precedes the occurrence of bronchiectasis.[1]

Fig. 182.

Cretaceous enlargement of a bronchial gland compressing the right bronchus, which is much dilated beyond the point.

When the changes have taken place in the tissues which are likely to give rise to it, any violent effort to distend the lungs, as in catarrh, bronchitis, or hooping-cough, may be the exciting cause. The three forms which Laennec[2] describes have been successively adopted by subsequent writers, though none have been satisfied with his rationale. In the first there is a solitary cystic dilatation—in the second, a series of distinct dilatations of a more or less circular form, commonly affecting bronchi of the third and fourth order; these two forms are essentially the same, and they generally present considerable attenuation of the dilated portions, while the intervening parts of the bronchi remain normal. Laennec's third form differs entirely from those just considered, and consists in an almost uniform or cylindrical expansion of a single tube, or an entire section of the bronchial tree. Here it is we meet with thickening of the parietes, tumefaction of the mucous membrane, which may be thrown into folds resembling those of the small intestine, and a proportionate increase in the subjacent fibrous tissue. When the dilatation affects the apex of the lung, it may proceed to such an extent as to resemble a tuberculous multilocular cavity. The perfect continuity

[1] Corrigan views the condition of the pulmonary tissues, giving rise to bronchial dilatations, as analogous to cirrhosis of the liver; he considers it the result of the formation of an unyielding fibrous tissue, to which the bronchi become attached, and therefore incapable of collapsing.

[2] See Laennec's Traité de l'Auscultation Médiate, &c., 1826, vol. i. p. 206.

of the mucous lining with that of the adjoining bronchi, and the smoothness of the tissues, will aid in determining the nature of the lesion, if there is any doubt. The explanation offered by Hope[1] of the mode in which bronchitis, the most frequent exciting cause of bronchiectasis, gives rise, is so satisfactory, that we give it in his own words: First, the air-passages are stripped of their epithelial lining in the ordinary manner; their canals becoming loaded in part with a mucous secretion, in part plugged with fibrinous exudation. This latter occurrence takes place chiefly within certain of the lesser twigs, occasioning a collapse of the adjunct air-cells. The space thus set free, is sought to be filled up by the expansion of the neighboring parts, giving rise, in the majority of cases, to emphysema; where, however, the collapse does not occur close beneath the surface of the lining, but at a greater depth, and near a larger bronchial tube, and when it comprehends a larger tract of pulmonary substance, the result is bronchiectasis; these circumstances do not, however, suffice for the formation of a bronchial cavity, the parietes of the involved bronchial tube must needs have previously suffered the changes pointed out by Stokes—namely, loss of elasticity in the longitudinal, and of contractile power in the annular fibres, with consequent incapacity on the part of either to resist the mechanical influence of forcible inspiration or of violent cough.

It appears from Dr. West's description, that in children the fusiform variety of bronchial dilatation is rarely, if ever found, while the cylindrical form is a common result of bronchitis. On one occasion, however, he saw a case in which, in addition to a general cylindrical enlargement of the tubes, many of them presented a marked dilatation, about half an inch from their termination the tube expanding into a cavity big enough to hold half a large nut. The lining mucous membrane presented an extraordinary degree of thickening.

Tubercular deposit in the bronchial tubes is an affection spoken of by Rokitansky alone among authors; he describes it, under the term of bronchial tuberculosis, as an infiltration of the mucous membrane with yellow, lardaceous, caseous matter, into which the former at last appears to be converted; the bronchial tube becomes enlarged, its channel finally obstructed by tubercle, while its fibrous sheath is infiltrated with lardaceous matter, callous and thickened. He states that it occurs secondarily to tuberculous abscesses, but that the primary form, commencing in the ultimate ramifications of the bronchi, and extending backwards into the larger tubes, is by far the more important form. The latter may be found unassociated with genuine pulmonary tubercle, and it may, by softening and breaking down of the bronchial walls, give rise to abscesses resembling those of vesicular tuberculosis; at the same time, the author admits that this mode of the formation of abscesses is infinitely less frequent than that commonly known. The primary form of bronchial tuberculosis, Rokitansky describes as most common in childhood, being usually associated with all the tuberculoses of other organs peculiar to this period of life, and especially with intense tuberculosis of the bronchial glands.

[1] Pathological Anatomy, Syd. Soc. Ed., p. 297.

In addition to the varieties of mucous, purulent, and hemorrhagic sputa, and to the foreign bodies occasionally introduced from without, to which we have already alluded, we occasionally find in the bronchial passages calcareous concretions, derived from obsolete tubercular deposits, bile or bile-tinged pus, derived from a fistulous opening through the diaphragm, communicating with the liver fragments of pulmonary tissue, recognizable by the elastic fibres shown under the microscope, and cysts. The latter are extremely rare, and are probably always derived from the pulmonary parenchyma, or the liver. We have not seen any case recorded of their formation in the bronchi. Hasse[1] states, that he has seen a pellucid vesicle, of the size of a hemp-seed, on the left vocal cord, and he mentions cases recorded by other observers of hydatids in the same region.

[1] Pathological Anatomy, Syd. Soc. Ed., p. 378.

CHAPTER XXVIII.

THE LUNGS.

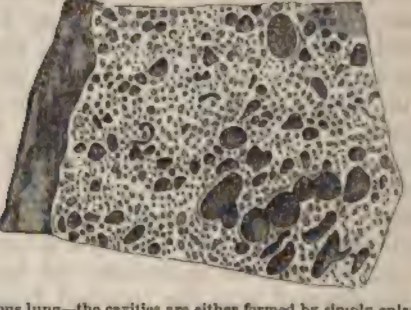
HAVING in the foregoing pages considered the diseases of the respiratory passages, we now arrive at the investigation of the morbid changes which occur in the lungs themselves; in the tissues aiding in the purposes of oxygenation, the ultimate vesicular terminations of the bronchi and the interlobular tissue. The bronchules, long before their termination, are deprived of their cartilaginous rings; these are reduced to mere flakes before they cease altogether, and all trace of them disappears, according to Messrs. Todd and Bowman,[1] in tubes of less than one-sixth to one-tenth of an inch in diameter. The tracheal muscular fibres are described by these physiologists as being continued even to the terminal bronchules, but instead of merely filling up the gap in the cartilaginous framework, they form a uniform layer encircling the canal, but excessively thin. Within the muscular layer is that of the longitudinal or elastic fibres; the ciliated epithelium and basement membrane of the mucous tissue descend into the terminal bronchules. The air-vesicle itself is described as formed of one coat only, resulting from a fusion of the elastic coat and basement membrane, and it is stated to contain no epithelium. Whether the latter point is correct or not, the extreme tenuity of the vesicular coat is an undoubted fact; nor could it be expected otherwise, when we consider that the part it plays is to prevent the air from being diffused into the interstitial tissues, and that the exchange of the oxygen of the atmosphere, and of the carbonic acid of the blood, could only be effected with sufficient rapidity through an infinitely delicate texture. We premise so much of the healthy anatomy of the lung, in order to render it evident that the difference in its texture would produce different morbid symptoms, as well as different post-mortem results, than those exhibited in diseases of the air-passages. While the latter possess their own peculiar secretions, which tend to modify or relieve the perverted action of their constituents, the effect of disease upon the true pulmonary tissues must be, to cause a more immediate interference with the vital functions, by altering the caliber of the air-reservoir from without or from within. The air-vessels themselves are solitary globular sacs, terminating a minute bronchule, and arranged along a larger bronchule like a bunch of currants, or presenting a corymbose or racemose appearance more analogous to a bunch of grapes. The vesicles are never angular or polygonal, until subjected

[1] Physiological Anatomy, chap. xxix.

to pressure resulting from some pathological process. When intravesicular effusion is limited to a few scattered vesicles, the globular appearance is only rendered more conspicuous; and in no case has it appeared to us that the human lung offers, in its ultimate terminations, any resemblance to the honeycomb arrangement seen in the lungs of the lower animals, and produced by a reduplication of the basement membrane, so as to form septa, projecting into the cavity of the vesicle.

The anatomical characters of the vesicular structure, and the little support it has compared with the denser tissues of the bronchi, easily explain the first morbid condition to which we shall advert—Vesicular Emphysema. Dr. Baillie, though unable to suggest the means of distinguishing the disease before death, was one of the first to show its true nature in the dead body. It consists essentially in a dilatation of

Fig. 183.



Portion of emphysematous lung—the cavities are either formed by simply enlarged air-cells, or by the coalescence of groups of cells.—St. Bartholomew's Museum, xiv. 11.

a larger or smaller number of air-vesicles, and may be produced by any cause exerting a great strain upon them. The effect is to diminish the specific gravity of the part affected, so as to render it more buoyant than the healthy lung tissue in water; to cause the lung to become less crepitant on compression, giving it a doughy or woolly feel, to prevent its collapse on the thorax being opened, and to render it more or less dry and exsanguine. The emphysematous portion, if superficial, projects above the surface of the unaffected part; and large bullæ may be visible on the surface of the lung, from the gradual obliteration of the intervesicular tissue, allowing several vesicles to unite. The loss of elasticity in the pulmonary tissue, whether primary or secondary, prevents the usual collapse of the lung at the period of expiration; the vitiated air is not expelled as it ought to be; hence arises a great want of oxygen, and the patient, on the slightest aggravating cause, is seized with a fit of asthma; the consequence of this permanent dilatation of the organ necessarily affects the thoracic parietes; if one lung only is affected, the corresponding side enlarges, and becomes less movable than its fellow; the adjoining viscera are more or less displaced; the intercostal and supraclavicular spaces swell out; the ribs, instead of slanting downwards, stand out horizontally from the vertebral column, and are almost immovable, giving to the thorax that barrel-shaped form,

which alone is considered indicative of emphysema. In horses, this is a very common disease, and constitutes the vice termed "broken wind," which veterinary surgeons state to be chiefly due to overworking after a full meal of green meat. Veterinary surgeons have observed that it is hereditary in horses,[1] which tends to confirm the like remark made by Dr. Budd, Louis, Hasse, and others,[2] as applied to man. Dilatation of the bronchi is a pathological condition frequently associated with vesicular emphysema, and may be attributed to the same cause.

Mr. Rainey, in one of his interesting papers in the *Medico-Chirurgical Transactions*,[3] has attempted to prove, that emphysema is the result of over distension of the air-cells, brought on either by their mechanical distension, or of the pulmonary membrane undergoing a fatty degeneration, which enfeebles it, and causes it to give way under the ordinary pressure of inspiration. Dr. Gairdner, as we shall see, denies either mode of production in the sense of Mr. Rainey, and only admits the occurrence of fatty or granular deposits in emphysematous parts as exceptional. The microscope confirms what is palpable to the naked eye, that the bloodvessels are very scanty. They become compressed in the interstitial tissue by the enlarging air-vesicles, and are entirely obliterated in the progress of the disease; hence, the emphysematous portion is exempt from those morbid affections which are associated with congestion or inflammatory conditions, such as hemorrhage, pneumonia, or exudative processes. It was formerly supposed that vesicular emphysema was mainly due to rupture of the interstitial membrane; we have shown that this is not the prevailing circumstance; still, actual laceration does occasionally take place. The largest dilatations are seen along the ulterior margins of the lungs, probably owing to these parts being least supported. The bullæ individual sometimes attain a very considerable size. If the dilatation of the air-cells is more generally diffused, the lung presents, as Dr. Baillie first observed, the appearance of the lung in amphibious animals.

It is the more important to be well acquainted with the features of this malady, as much may be done to anticipate and prevent it, while we have little control over it when it is established; the advantage of the study of pathological anatomy, as an illustration of morbid processes in the lining, is rarely shown in more marked instance than here: the knowledge of the actual lesion giving rise to the phenomena of asthma, if not in all, yet in the majority of cases, is a warning to those who, from indolence or carelessness, are too prone to be satisfied with attributing spasmodic symptoms exclusively to deranged nervous action. The credit of offering the first rational explanation of asthma, and its connection with emphysema, is due to Dr. Floyer,[4] though the priority of

[1] See Mr. Youatt's work on the Horse, art. Broken Wind.

[2] Med.-Chir. Trans. vol. xxiii. p. 37.

[3] Vol. xxxi. p. 297.

[4] In his little work on Asthma, published in 1698, Dr. Floyer says: "The broken wind results from the rupture or dilatation of the bladders of the lungs, by which the air is too much retained in the bladders or their interstices, and thereby produce a permanent flatulent tumor in the whole substance of the lungs. It is not easy to explain the production of a permanent flatulent tumor in the lungs by a strain in running, but by supposing the bladders of the trachea too much distended, and the muscular fibres which constringe them

the discovery is commonly attributed to Laennec. The views of the latter, in regard to the production of the disease, though difficult of comprehension, have long been accepted by the profession. He conceives that any obstacle to the discharge of air from the lungs may give rise to it; he especially attributed it to "dry catarrh;" in early life, hooping-cough, by inducing an over-repletion of the air-vesicles, and a great strain upon them; swellings of the bronchial glands, by compressing the bronchi, frequently give rise to vesicular emphysema. We have stated that Laennec's view of the manner in which emphysema is produced, is difficult of comprehension, because a plug that prevents the exit of the air is likely to prevent its admission; and although a valve-like action may, by a curious combination of circumstances, be induced so as to be followed by such an effect, the structure of the bronchial tree forbids our assuming its frequent occurrence. There are other still more forcible arguments against Laennec's theory, which have been put forward by Dr. Gairdner.[1] He remarks that the theory of Laennec, which ascribes emphysema to mucus in the bronchi and accumulation of air behind the obstruction, is vitiated by the ample proof which now exists, that obstruction of the bronchi has precisely the opposite effect, giving rise to voiding of the air-cells and collapse of the lung; he rejects the idea that emphysema is dependent upon forcible expirations as utterly untenable. Dr. Gairdner has found that emphysema never occurs unaccompanied by pulmonary collapse, or by one or other form of pulmonary atrophy; the greater volume of the emphysematous lung depending, not upon increase of tissue, but mere distension. He denies that it is ever preceded by any altered condition or diminished resistance in the walls of the air-vesicles; he has usually found the bronchi leading to affected parts entirely unobstructed; and he concludes that emphysema is a lesion occurring from mechanical causes, in those parts of atrophied and collapsed lungs to which it has the most free access; in other words, it is produced by atmospheric pressure in the comparatively sound portions of such lungs. According to Dr. Gairdner's theory, the increase of volume of the emphysematous lung is supplementary to the diminished volume of those parts from which the air is excluded; but the real effect is, that while the surface of the breathing membrane is extended, its physiological capacity of aeration is much diminished, so that the individual comes to labor under two morbid conditions instead of one. Another form of emphysema is that to which the name interlobular has been applied, and which is analogous to the emphysema occurring on the surface of the body; like this, it consists in an effusion of air into the cellular, or rather, as applied to the lung, into the interstitial tissue, and is due to a laceration of the bronchial tubules or the air-vesicles. It generally induces a puffy swelling under the pleura, and is commonly associated with a subcutaneous extravasation of air, affecting the chest, neck, and head. Emphysema, in its turn, like every disease which impairs the circulation

in expiration, thereby over-stretched and made unfit to express the air afterwards; so that these bladders retaining more air than is usual, the substance of the lungs must appear always inflated."

[1] British and Foreign Medico-Chirurgical Review, April, 1858, p. 452.

through the lungs, is liable to induce dilatation of the right side of the heart.

We must be careful to distinguish the two forms of emphysema just alluded to from one another, as well as from the post-mortem evolution of gas, which may produce similar appearances. In the latter instance, there are general indications of decomposition to guide us; and we shall find that the bladders of air that distend the pleura may be easily removed by pressure, and the accumulation pushed aside more readily than when we have to deal with a pathological product. Nothing, in fact, is more difficult than to free an emphysematous portion of lung from air; even in drying it slowly, the diseased cells, as Hasse observes, retain their air, whilst the undilated cells dwindle down into a hard and almost solid mass. The determination as to the nature of the emphysema may be of importance in forensic medicine, in determining the question as to the employment of violence in causing the death of an individual.

ATELECTASIS PULMONUM.

Diametrically opposed to the condition which we have just considered, is one which is peculiar to early life, and frequently has been confounded with the results of pneumonia. It consists in the permanence of the fœtal condition of the lungs; the vesicular structure either not being properly developed, or the infant not possessing sufficient force to expand the thorax, and cause the dilatation of the breathing apparatus. It was first observed, and duly described, by Professor Jörg,[1] under the name of Atelectasis Pulmonum;[2] who found that it occurred chiefly in full-grown infants of very feeble powers, or in such as had been born prematurely, and were, therefore, not in a condition to dispense with the placental circulation. The inferior portions of the lungs are most liable to present this state; it occurs in patches, which offer a darkened color, do not crepitate on compression, and offer a smooth surface on section. The affected part sinks in water. The part or lobule that has not undergone the due expansion is below the level of the surrounding dilated lung tissue; hence, a lung affected in this manner does not fill the cavity of the thorax as the lung that has performed its functions; nor does it, on section, discharge a frothy sanguineous serum like the latter. Atelectasis is not necessarily fatal; it is probable that in many instances it is entirely overcome, as the child acquires vigor, or that, while a few lobules remain in a permanently contracted state, the remainder of the lung suffices for the purposes of aeration. We frequently, in adults, meet with puckerings of the surface of the lung, without any trace of inflammation, which may perhaps be set down to this congenital state of the tissues; or, possibly, the small nodules of fibroid or calcareous matter, which we often find equally without appreciable recent lesion to which they could be referred, may be due to the same cause, as we know them to favor parts in which there is an arrest of development. Dr. West observes

[1] De Pulmonum Vitio Organico, &c., Lips. 1832, and Die Fötuslunge im Gebornen Kinde. Grimma, 1835.

[2] Etymology—ἀτελής, imperfect; ἔκτασις. expansion.

that if air be blown into a lung, some lobules of which are not duly expanded, it will permeate the collapsed air-tubes; the pulmonary vesicles will, by degrees, become distended, and the solid lobules rise to a level with the rest of the lung, acquiring the same color and consistence, and, like other parts of the organ, will float in water. A single inflation, however, is by no means sufficient to render this change permanent; but, the moment the tube is withdrawn, the air will escape, and the lobules recently distended will again collapse and sink below the rest of the lung; their color, too, will become dark, though less so than before. In conjunction with imperfect expansion of the lung-tissue, we invariably find the foramen ovale and the ductus Botalli still open. The pathology of the affection, which we have attributed mainly to a mechanical defect, has been explained by French writers, who have termed it carnification, as the result of pneumonia; but, although pneumonia may supervene in an atelectatic lung, the characters of the two diseases are sufficiently distinct to be discriminated on a careful examination. We meet with a condition analogous to infantile atelectasis in advanced age; we find the apices of the lungs converted into a dense melanotic mass, in which we are unable to trace tubercular deposit, while there is an obliteration of the vesicular structure, and apparently also of the bloodvessels. The tissues are converted into a viscid, tenacious mass, deprived of all air. Under the microscope, we see an almost homogeneous membrane, through which the carbonaceous deposit is scattered irregularly, with only here and there a trace of the circular fibres of the lung. In the cases which have fallen under our own observation, there was a thickened pleura, forming a cap over the apex, which must, by its compression, have contributed much to the obliteration: whether any pneumonia had aided in producing the result is doubtful; our impression certainly is against this view, the excessive deposit of carbonaceous matter associated with the large pleuritic exudation in the confined post-clavicular region of the thorax appearing adequate in itself to account for the effect produced. Another question is, whether fatty degeneration affecting any of the tissues was an element in the process; possibly, the deposit of carbonaceous matter in excess may be viewed as the result of a process allied to fatty degeneration: we have not ourselves observed such an evolution of oil as would justify the assumption that the main feature of the disease belongs to this class.

Another form of atelectasis, which Bailly and Legendre first pointed out, and which has been well illustrated by Dr. West, is that which occurs after respiration has once been fairly established, and is the result of an interference with the mechanism of respiration. It is this form more particularly which Valleix, and Rilliet, and Barthez, under the term of carnification, have attributed to lobular pneumonia; the affection being limited to a single lobule or to a cluster, forming a hard, compact mass, surrounded by the normal tissue. When the affected part is inflated the vesicles distend, and thus show that there is no inflammatory effusion. In a child, whose case is related by Dr. West, there was no evidence of disease until the age of nine months, although she had not thriven well, and had become pigeon-breasted. She then lost flesh rapidly, and began to cough, without having had any previous

catarrh. Her case seemed to be one of bronchial phthisis. Four days before death she became suddenly oppressed, and the cough more severe; the dyspnœa increased, while the cough became less frequent. A few hours before death the lips were quite livid; she breathed from eighty to eighty-six times a minute; the abdominal muscles acting most violently, but the chest being scarcely at all expanded. No tubercle was found in any organ after death, but large portions of both lungs presented the undilated condition, which disappeared entirely on inflation; the bronchi were pale, and contained very little mucus; the right side of the heart was greatly distended with coagulated blood, which its thin, pale, and flaccid substance, had evidently been unequal to propel with the requisite vigor.

There are consecutive conditions of the lungs which may respectively be mistaken for emphysema or atelectasis. Thus, after long-standing disease of one lung, impairing its functions and diminishing its capacity, we find its fellow taking on a vicarious action, and expanding so much as to displace adjoining viscera; a point of importance in forming diagnoses of diseased states of the thoracic contents. On opening this side of the thorax the lung may appear too large for its cavity, and induce the assumption of an emphysematous condition. The history of the case, the examination of the lung, and the shrunken, contracted, bound-down, and atrophied condition of its fellow, will determine the real nature of the case. Whether in this instance there is an actual new formation of pulmonary vesicles, it is difficult to ascertain; Rokitansky is of opinion that such an hypertrophy of the lungs is due to a dilatation of the air-cells, with a simultaneous augmentation of their tissues; that it does not consist in an increase in the nùmber of the air-cells, but in their dilatation, the increased thickness of their walls, the enlarged caliber of their walls, and in the development of their vessels. There can be no doubt that the changes that take place are intended to increase the powers of aeration of one portion of the breathing apparatus; an increased thickness in the walls of the air-vesicles can scarcely facilitate oxygenation, unless it is by a reduplication of the basement membrane into the breathing cavity, so as to afford a larger surface for the capillaries to ramify upon. The special circumstances under which the secondary enlargement and contraction of the lungs take place, will be a subject for further consideration at a subsequent page, when we come to discuss the pathological states of the pleura.

ŒDEMA PULMONUM.

Before investigating the inflammatory conditions of the lungs, we are required to devote some consideration to a morbid state, which we frequently meet with in the dead body, and which, since the attention of the profession was first especially directed to it by the researches of Laennec,[1] has been known by the term of Œdema Pulmonum. It consists, as its name implies, in a serous infiltration of the interstitial por-

[1] De l'Auscultation Médiate, vol. i. p. 349.

tion of the pulmonary parenchyma. It causes a puffiness of the organ, which pits more or less on pressure, has lost its natural crepitant sensation, and does not collapse when the thorax is opened. The œdematous lung is characterized by pallor and anæmia, and when cut into discharges an abundance of clear, limpid serum, in which the comparative absence of air-bubbles is characteristic. It occurs in connection with, and as a result of, a great variety of debilitating diseases, and rarely presents an idiopathic character, though it is sometimes observed in this form, and may then be termed serous apoplexy of the lungs. Pulmonary œdema very commonly supervenes immediately before death; and has been attributed to the extinction of the nervous power of the vagi, in consequence of the experiments of Müller, which have shown that the fatal effect of dividing these nerves in the neck, is mainly due to the infiltration of the lungs and air-passages with serum. Both lungs are generally affected to the same extent, nor is the œdema necessarily confined to the posterior portions, even where it is only partial. A peculiar fact, remarked by Hasse, is, that where, in general dropsy which proves fatal, the one lung is found universally adherent to the pleura, and the other not, the former is œdematous, and the latter compressed by hydrothorax. Laennec states that pneumonia induces a great proclivity to the production of pulmonary œdema during the period of convalescence: this may have been partly due to the excessive depletion formerly in vogue, for pulmonary œdema appears to occur so frequently, and so much in the ratio of the general anæmia of the individual, that we see no reason for assuming a special tendency in one disease to produce it. The lax texture of the lungs, like that of the superficial cellular tissue, necessarily favors a serous effusion under such circumstances.

PULMONARY CONGESTION.

This spongy texture of the lungs, coupled with the fact that these organs contain a larger quantity of blood, in proportion to their size, than any other organ of the body, renders them peculiarly liable to the various forms of congestion—a tendency which is enhanced by the relation existing between the pulmonary and systemic circulation. Most of the various causes of death, while inducing an arrest of the circulation, give rise to an accumulation of blood in the organs of respiration. Nysten's experiments have shown, that the contractile power of the right side of the heart continues long after the irritability of the left side is extinguished; and the effect of maintaining artificial respiration in cases in which death from a lesion of the nervous powers is to be apprehended, further demonstrates the great share taken by the lungs in the production of death. The elasticity of the arteries also exercises some influence in producing an engorgement of the lungs, at the moment of, and immediately after death, by propelling their contents into the venous system, and thus overcharging the right side of the heart. Nor must we forget that, in long-standing debilitating disease, whether a specific fever or an adynamic condition, resulting from other disorganizing processes, respiration is carried on with little vigor, while the mus-

cular tone is reduced to a low ebb, so that both causes conspire to retain the blood in the pulmonary tissues. Hence, in estimating the pathological changes in the lungs connected with the actual disease, to which death has been attributed, we must be very careful in distinguishing between the effects of the dying and death itself, the secondary products of debility and dissolution, from the changes attributable to active disease. Both, however, often pass imperceptibly into one another, with gradations, which only confirm the view that disease is, in itself, incipient death. And though we may lay down rigid classifications of the modes of dissolution, and we may occasionally meet with types corresponding to our scientific arrangement, still, as Dr. Williams observes, in his admirable *Principles of Medicine:* "In the slower dissolution by which diseases generally prove fatal, all functions and structures are more or less involved, and life in all is dwindled down to so slight a thread, that, when it breaks in one, others scarcely retain it long enough to enable us to say that death begins distinctly in any part." Still, whether we can trace the death to asthenia or apnœa, coma or paralysis, the prevailing effect is to induce those symptoms to which we have above alluded in the lesser circulation.

It is not within our scope to dwell upon the treatment of disease, but we may be allowed to urge these facts as of the utmost importance in connection with that department of medicine, inasmuch as they demonstrate the necessity of, under all circumstances, attending to the state of the pulmonary circulation, and removing all avoidable sources of local embarrassment, while we stimulate the general forces to carry the patient through the valley of danger to the pleasant heights of recovery and health. The congestion which belongs chiefly to the causes first alluded to, is most liable to affect the posterior and inferior portions of lungs; after death, as in the debility resulting from disease, the blood follows the physical law of gravitation, and sinks to the lowest point it can gain. If there be no concomitant inflammatory changes, the congested portion presents a dark red color, and, though firmer than the more bloodless anterior part, still crepitates under the finger, and floats in water. The color is almost uniform, and the line of definition between the congested and the non-congested portion is tolerably defined. The pleural surface of the engorged portion presents a corresponding violet tint, which sometimes is more or less circumscribed at single points. The depth of the color varies somewhat in different diseases; and in very anæmic cases, especially in those associated with general dropsy, there is more or less serous effusion with the sanguineous congestion. In a medico-legal point of view, congestion of the lungs may become a question of life and death; thus, in the recent trial of Mr. Kirwan, in Dublin, the conclusion that his wife's death was due to violence, which has since been shown to be erroneous by the highest authority in medical jurisprudence in this country, Dr. Taylor,[1] was based mainly upon the fact of the lungs being congested posteriorly. This was the main fact upon which the medical witness, Dr. Hatchett, relied, in proof of death having been brought about by drowning; we know that it may be the result

[1] Dublin Quarterly Journal, Feb. 1858.

of post-mortem changes, and, as Dr. Taylor observes, "it is not of the least value as medical evidence of drowning, unless observed soon after death, and unless attended with other appearances, which, upon the assumption of death by drowning, or by some other form of asphyxia, ought always to accompany it."

But recently, a lad was examined at St. Mary's Hospital, who was brought in asphyxiated by drowning; he was, in fact, dead at the time, but still some efforts were made to restore him. The post-mortem examination showed none of the visible signs commonly attributed to drowning, and there was no congestion of any of the viscera.

It is doubtful whether there are any means of determining whether hypostatic congestion has occurred after death or within a few days of dissolution. We know that in full vigor the blood is not disobedient to the laws of gravitation, as we may easily ascertain by allowing our arm to hang down and then raise it into a vertical position, or by elevating our feet above our head; therefore, it is not surprising that, in the recumbent position, as the powers of life fail, the blood should gravitate to the posterior portion of the lungs. If the congestion is confined to one lung, or to the anterior parts of the lungs, we may safely attribute it to morbid processes; and if there are any other traces of inflammatory action, to which we shall advert further on, we may equally set down the congestion to a pathological cause.

Hypostatic congestion is closely allied to the disease which has been termed *pneumonie des agonisans*, by Laennec, and has been fully described by Mr. Erichsen[1] as the congestive pneumonia, to which the majority of deaths following capital operations are due.

PULMONARY APOPLEXY.

The most formidable phenomenon to which congestion of the lungs gives rise, is the disease to which, from the earliest times, the term of pulmonary apoplexy has been applied. It is distinct from the hemorrhage that occurs from the bronchial mucous membranes, either owing to an adynamic state of the blood or to active congestion, both in the symptoms it produces during life, as well as in the post-mortem appearances. The seat of pulmonary apoplexy is the parenchyma of the lung, and, most probably, with few exceptions, only the intervesicular tissue, for it is rarely associated with hæmoptysis, which we should expect if the effusion took place into the air-vesicles themselves. The apoplectic spot may be felt before the lung is cut into as a globular mass, of greater density than the surrounding tissue, and, if near the surface, its darker color also attracts attention. On section, we find a dark red, almost black, homogeneous, circumscribed spot, varying in size from a pin's head to an orange, of the appearance and consistency of damson cheese, bounded by tissue, which is comparatively healthy both in color and consistency. The only interruption to the uniform color that is met with, is that caused by the dividend bronchules, which are less dark

[1] Medico-Chirurgical Transact. vol. xxvi. p. 29.

than the surrounding parts. The more recent the hemorrhage, the more defined the outline; while, if the patient has survived the immediate shock, the margin of the spot fades away, owing to incipient absorption. Thus we have seen, as in the patient from whom the accompanying drawing was taken, several spots, evidently varying in their date. The breathing capacity of the part is entirely destroyed; it contains no air,

Fig. 184.

Pulmonary apoplexy, occurring in a man aged 53. There were several apoplectic masses, exhibiting a deep purple, almost black hue, and causing an homogeneous solid appearance of the part affected, as shown in the section.

and when scraped, only yields a dark, thick, bloody fluid, in which the microscope detects nothing but blood-corpuscles and some pulmonary debris. If the margins of the clot be scraped and examined, we may find exudation corpuscles, varying in size from $\frac{3-7}{5000}$ of an inch, showing that some irritative action and organic change are going on.

The base of the lungs is the part most commonly affected, and there may be one, two, or three isolated apoplectic spots. Laennec, who viewed pulmonary apoplexy and hæmoptoic engorgement as modifications of the same disease, considered hæmoptysis as a symptom equally of both, and was of opinion that the former not unfrequently terminated in resolution and recovery. Without disputing the possibility of the absorption of an apoplectic clot, it certainly appears that there is an essential distinction between bronchial and parenchymatous hemorrhage, which we must explain by a preliminary alteration, as yet not sufficiently understood, in the proper lung tissue. This is the more probable when we consider that the hæmoptysis which is the harbinger and concomitant of tubercular disease, rarely, if ever, presents the lesion denominated pulmonary apoplexy, and that the latter is frequently met with in the dead body without its presence having been manifested by hemorrhage during life. Louis[1] states that, during the epidemic yellow fever, which occurred in Gibraltar, in 1828, pulmonary apoplexy was very frequently found in the victims to the disease, in none of whom hæmoptysis had taken place; on the other hand, he had never, in phthisical patients who had died during, or shortly after, attacks of hæmoptysis, met with ap-

[1] Researches on Phthisis, Syd. Soc. Ed. p. 168.

pearances resembling those of pulmonary apoplexy. We are, therefore, in every way justified in looking upon the two affections as essentially distinct from one another.

Pulmonary apoplexy occasionally gives rise to hemorrhage into the pleural sac, from a laceration occurring in the pleura; this will probably be owing to the same process of disintegration, which permitted the apoplectic effusion in the first instance.

The interstitial form of pulmonary hemorrhage may be due to various predisposing causes, that affect the constitution of the blood, or of the tissues, or both together; while the exciting cause is most commonly to be found in a morbid condition of the heart and great vessels, and more particularly in hypertrophy of the right ventricle. About two-thirds of the cases on record have exhibited some lesion of this kind; still, as Hasse very justly remarks, a preternatural condition of the pulmonary texture appears always to precede, while a chemical analysis of the blood would probably exhibit a scorbutic diathesis or an hydræmic character. It has been observed that drunkards are prone to be affected with pulmonary apoplexy.

The secondary processes to which apoplectic spots of the lungs are subject are, a gradual absorption of the blood effused, suppuration and abscess, or isolation by the formation of a cyst; none of these processes, however, extend far, and with regard to the last, no satisfactory proof is extant of its occurring at all, beyond an imperfect observation by Bouillaud.[1]

[1] Archives Générales de Médecine, vol. xii. p. 399.

CHAPTER XXIX.

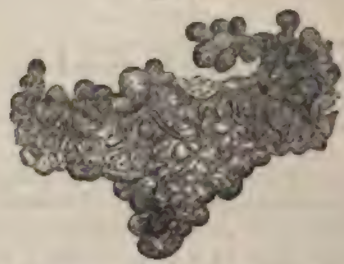
PNEUMONIA.

INFLAMMATION of the pulmonary tissue is commonly assumed to present three stages, which we may trace in regular succession in the patient, or which we find coexisting at various portions of the lungs in the same dead subject. The first stage, that of congestion, we have already considered; its situation, its effect upon the cohesion of the tissues, the coexistence of other inflammatory changes, and the history of the case must assist us in determining its character, though it is often difficult to be certain of its nature. The general effect of acute inflammation, in altering the cohesion of the tissues, is a point of considerable importance; it particularly affects, as Sir Robert Carswell has pointed out, the uniting cellular element, and may thus demonstrate the previous existence of inflammation, though the redness and vascularity have disappeared, or but faintly mark the degree of alteration which the disease has effected in the process of nutrition. This general law is compatible with the observation that the second stage of pneumonia, or hepatization, is accompanied by a state of solidification; for, as the first-named author remarks, though the tissues feel harder than natural when compressed, the diminution of cohesion which has taken place between their anatomical elements is rendered conspicuous by the facility with which they are penetrated, broken down, or crushed.

In doubtful cases the microscope would aid us, by determining the presence or absence, in the congested portion, of exudation-corpuscles, which we find where the naked eye fails in distinguishing the existence of inflammation, and which, at all events, show that some organic metamorphosis of the vital fluids is taking place, not consistent with the ordinary physiological changes. The confines of the first and second stages merge into one another, and are often difficult to define. In the second, the stasis of the blood becomes more marked, the specific gravity of the pulmonary tissue increases, the overcharged vessels relieve themselves by fibrinous exudation into the interstitial tissue, and by slight hemorrhage into the air-vessels, which, mingling with the bronchial secretions, gives rise to the pathognomonic rust-colored expectoration of pneumonia. This stage has received the name of hepatization, owing to the increased density of the parenchyma causing the affected portion of the lungs to resemble a piece of liver. M. Andral, who considers the softening process to predominate, prefers the term ramollissement rouge, as applied to this condition. It is well to remember that both designations are used indiscriminately for the same morbid condition.

The color of the affected part is of a dark red, which is more or less venous or dusky, in proportion to the type of the inflammation; the crepitant character of the tissue is fast disappearing; the lung, on section, has lost that light spongy appearance peculiar to it in health, and

Fig. 185.



Lung in a state of red hepatization; the air-cells are filled with corpuscular fibrin or exudation-matter, and are surrounded by enlarged and congested vessels. Magnified twenty diameters. (Bayle's Granulations.) From a man æt. 66, who had double pneumonia.

but little frothy red fluid exudes from it. On the pleural surface, instead of the slate-colored, marbly hue of the normal state, we find a more uniform, dusky-red color, scarcely broken by the interlocular septa. At this period, as Gendrin first pointed out, repeated washing and continued maceration fails to restore the natural color of the tissues, which they recover, under such a process, if the redness is due merely to congestion. Before that exudation and general infiltration have taken place, which constitute the succeeding stage of pneumonia, we find that, on breaking up a portion of hepatized lung, the surface is studded with small pinky granulations, which are identical with the pulmonary granulations of Bayle. This author, and some of his successors, looked upon them as the first stage of tubercular disease; but, as Andral has satisfactorily shown, they are a product of inflammation, and must be considered as an agglomeration of a few hyperæmic vesicles.

Though the specific gravity of the hepatized lung is considerably increased, the dimensions of the organ are not necessarily augmented; occasionally, however, the organ is shown to be enlarged by the indentations left on its surface by the ribs.

In the present state of our knowledge of inflammatory processes generally, and of the form which they assume in the lungs especially, it is impossible, with certainty, to show the minute changes which take place in this stage of pneumonia. That the parietes of the pulmonary vesicles should themselves thicken, consisting as they do of basement-membrane only, is, at least, not proved, and, though given on the exalted authority of Andral, he fails to state the actual grounds for the view which he takes. That some change occurs in the solids as well as the fluids of the inflamed part, that the chemical and physical properties of both undergo an alteration, that their mutual affinities differ from those of health, is manifest from the phenomena of disease, but the ultima ratio yet remains to be discovered. Andral's theory of the pulmonary granulations being due to injection and tumefaction of the

parietes of the air-vesicles, is not consonant either with theory or observation; for it is not difficult to trace the deposit within a cavity, nor is it intelligible, without actual demonstration, how swelling of a membrane could produce such uniform nodules without any anatomical reason to assign for such regularity. We regard the second stage of pneumonia, that of red hepatization, as a perpetuation of the congestive stage, with incipient exudation into the parts surrounding the vessels of the albuminous constituents of the blood; the exudation takes place both into the interstitial and into the inter-vascular spaces, according to the laws regulating the exosmosis of inflamed vessels; the microscope exhibits a vast congeries of gorged vessels permeating the pulmonary structure and surrounding the air-vesicles; while the exuded fluid shows a granular blastema, blood-corpuscles, and exudation-cells, which are, probably, of an oily or albuminous character.

These exudation-cells, here, as elsewhere, are the product of organic or bio-chemical changes, and not, as Hasse[1] would seem to imply, the mere effect of chemical agents applied on the table of the microscope. They can be traced in the various stages of formation from the granular corpuscles to the agglomerate corpuscle and the perfect cell immediately on removal of the fluid from the body; and their character is essentially the same, as we have elsewhere shown, in the most various situations.

The different degrees of congestion of the inflamed portion produce a mottling of the surface; the congestion itself may be circumscribed with a definite line, or it fades off gradually into the healthy tissue. As the second stage progresses, marbling of a different kind occurs, which is due to the gradual disappearance of the coloring particles of the blood, the absorption of the blood itself, and the substitution of a fibrinous deposit, or pus. We now enter into the third stage of pneumonia, or that of gray hepatization. The term well denotes the appearance of the affected portion; it is entirely consolidated and deprived of all air; it presents a grayish or grayish-yellow color, which is only varied by the almost linear remains of the compressed bronchules, and the pigmentary matter scattered through the lung tissue. The general condensation of the tissues necessarily, also, involves the bloodvessels, and arrests and prevents the circulation; so that the further changes must be mainly due to extravascular metamorphoses. The parenchyma becomes gradually softer, and the more straw-colored and paler its hue, the more fully the suppuration process is established, and the more friable the tissue becomes. A purulent fluid now exudes on pressure. At the commencement of this stage the air-vesicles may be isolated in the shape of gray granulations, of a globular form, containing an opaque granular matter. The microscopic characters of gray hepatization, at this period, have erro-

Fig. 186.

Microscopic characters of the contents of an air-vesicle in gray hepatization, consisting of granular matter, pus-corpuscles, exudation-cells, and cylindrical epithelium.

[1] Pathological Anatomy, Syd. Soc. Ed. p. 211.

neously been stated to be those of suppuration only; but true pus-cells are by no means the predominant forms seen under the microscope; the corpuscles that we have found to prevail were much larger than pus-cells, varying from $\frac{3}{5000}$ to $\frac{5}{5000}$ of an inch, presenting a sharp outline, and containing one or more glistening granules or globules, resembling oil. These are seen surrounded by a granular stroma, which also contains pus-corpuscles, free oil, cylindrical epithelium, and some forms resembling fibroid cells. The great rarity of actual abscess occurring in the lungs, compared with the frequency with which pneumonia reaches its third stage, would alone indicate, that this part of the process does not consist simply in suppuration; and we would explain it by the double exudation, which takes place into the vesicles and into the interstitial spaces, the one continuing somewhat, though remotely, under the influence of the atmosphere, the other being entirely beyond its action. The condensation that takes place in the tissues in pneumonia, and the form of the deposit, is of a very different character from that occurring in tubercular disease, and would alone serve to prove that the exudation takes place both within and external to the air-vesicles. The consolidation of pneumonia does not produce any material change in the form of the vesicular structure, which continues to present its globular form even in the third stage; now, if the exudation took place exclusively within the vesicles, their mutual compression would produce polyhedral forms, while the exclusive effusion external to them would not only obliterate them, but entirely destroy all trace of them; as it is, we continue to trace their form, because the extra and intra-vesicular effusions being tolerably uniform, the pressure is equable, and no material alteration in the form of the air-cells takes place. This is not the case, as we shall see, in tubercle, where the effusion is limited to the intra-vesicular spaces.

When the third stage of pneumonia advances to a fusion of the inter-vesicular septa, and an entire breaking down of the tissues, all trace of the normal structures disappears, and we only find a confused mass of pulmonary debris, pus, and ichorous sanies. We then have to deal with genuine pulmonary abscess.

The parts most liable to idiopathic pneumonia are the inferior portions of the lungs, while the upper lobes and apices of the lungs are rarely affected with pneumonia, except in connection with tubercle. Louis states that he has constantly found pneumonia affecting the upper and anterior part of the lungs, without a trace of the disease existing posteriorly, to be tuberculous; and he lays it down as a rule that this localization of pneumonia may lead to the diagnosis of tubercular disease previously undiscovered. This is undoubtedly correct in the main, but as Dr. Watson observes, it is probably exaggerated; and exceptions to the rule, regarding pneumonia, are met with, as well as in reference to the ordinary site of tubercle. Indeed, the numbers given by Andral would almost destroy the validity of the law altogether; for though they yield the preponderance to the lower lobes, they seem to show a much greater proclivity in the upper than they are commonly supposed to possess; in eighty-eight cases of pneumonia, he found it limited to the

lower lobes in forty-seven; in thirty in the upper lobes, and in eleven the entire organ was inflamed.

The general tendency of pneumonia is to spread from below upwards; and for this reason we commonly meet with the several stages in the same lung; the base to a greater or less extent presenting the gray hepatization—red hepatization affecting the adjoining portion next above the former—while the upper lobe offers more or less pneumonic congestion. The smaller bronchi of the affected part are not, as has been stated by several authors, invariably affected at the same time; we often see them meandering, as white rivulets, through the inflammatory mass; and they occasionally appear to possess a repellent power, and to form a line of demarcation between two parts that are unequally affected. A curious exception is mentioned by Rokitansky, in which, owing to bygone pleuritic effusion, the base of the right lung had not recovered its pristine elasticity, and where the entire lung was in a state of red hepatization, with the exception of the apex, the anterior margin, and the base, which latter is scarcely ever found exempt. It is equally exceptional to find pneumonia limited to the central portion of the lungs, though such cases undoubtedly occur; hence, one of the most ordinary complications of the disease is with pleurisy; for which reason some authors prefer the compound term pleuro-pneumonia, to the separate names pleuritis and pneumonia.

According to the unanimous testimony of all observers, the right lung is more frequently affected than the left, while double pneumonia is less often met with than either; the analysis of two hundred and ten cases of pneumonia by Andral,[1] yielded the following results:—

The right lung alone was the seat in	121
The left do. do.	58
Both together do. do.	25
The seat undetermined in	6
Total	210

The relation is somewhat, though not essentially, altered by the larger numbers collected by Sir John Forbes; he finds that in 1131 cases—

The right lung was affected in	562
The left in	333
And both together in	236

The pneumonia, as we have described it, is the form which has received the name of lobar pneumonia, as affecting entire lobes, in contradistinction to the lobular form of pneumonia, in which it is limited to individual lobules scattered through the healthy lung tissue. It is, in fact, a partial pneumonia, which, from some unknown cause, does not spread beyond the point of primary lesion. Small spots, with a scalloped outline, presenting the various stages of congestion, of red and of gray hepatization, are found on section scattered indiscriminately through the organ. It is peculiar to infant life, and is probably connected with the same imperfect development of the vesicular tissues, which we have already found to constitute an important disease of early

[1] Clinique Médicale, vol. iii. p. 470.

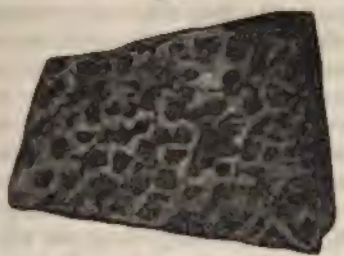
childhood. In many instances there is no doubt that the two conditions have been confounded. This cannot, however, be the case when we find the lining studded with small, yellow spots, satisfactorily indicating the third stage of pneumonia. Thus, in a child of one year and a half old, which was under our own observation, and from his ninth month had been subject to repeated attacks of broncho-pneumonia, we found the whole of both lungs studded with small purulent deposits; on the left side, where the softening process was furthest advanced, pus readily exuded on pressure; there were no cavities, nor was any tubercle discovered. The latter point, the absence of tubercle, is one of importance in determining that these purulent accumulations are not vomicæ, but due to a simple inflammatory process, independently of other characters that mark tubercle. The disease is stated by writers to result from phlebitis, accidents, and operations; but we shall have occasion to see that secondary abscesses and the form of pneumonia peculiar to individuals who have been operated upon, differ materially from lobular pneumonia.

According to Rokitansky, the character of ordinary pneumonia is essentially allied to croup, on the ground that the exudation takes place into the air-cells, and not into the interstitial tissue; this author distinguishes two forms of exudation in pneumonia, the one taking place into the cavity of the vesicles, the other interstitial. This classification is not borne out by what we see in the dead subject; and we are of opinion that the inflammatory effusion takes place into both at the same time. Our means of analysis are scarcely sufficiently advanced at present to determine whether it predominates in one or the other occasionally. Moreover, the character of croup is so peculiar, and the term generally understood to be applied to a form of inflammation of certain mucous membranes, so distinct from the inflammatory processes to which they are ordinarily exposed, that we are only likely to produce confusion by applying it to processes which, theoretically, we may consider allied to it. In the present instance there is not, however, even any theoretical presumption in favor of Rokitansky's view, for there is no relation between the occurrence of pneumonia and angina membranacea; the latter is essentially a disease of childhood, the former is the property of all ages; a peculiar habit tends to produce the one, certain seasons and places also create the proclivity to it; while in the other, inflammation of lungs, we do not trace the agency of the same influences. The description of the minute changes recurring in pneumonic exudation, which we have given, also tends to disprove the existence of an analogy, much less an identity, between the two processes.

The varieties of pneumonia which are spoken of by authors, as differing from ordinary acute pneumonia, are all characterized by presenting a more asthenic and less acute type, than the ordinary form which we have just discussed. The hypostatic pneumonia of Piorry, typhoid pneumonia, congestive pneumonia, the bilious, and the erysipelatous form of Riverius, and older writers, the senile pneumonia described by Hourmann and Dechambre, are all forms of asthenic pneumonia, occurring in subjects debilitated by other diseases, in whom the lungs are attacked by a low, creeping, sneaking inflammation. The congestion

is of a more venous character, causing a dusky brownish violet tint; the physical law of gravitation exerts a strong influence upon the blood in the thorax, and the posterior portions are predominantly affected;

Fig. 187.



Pleural surface of a portion of splenified lung, affected with typhoid pneumonia, from a female æt. 25, who died of typhoid fever. The lung closely resembled the spleen in consistency, was of a brownish red hue, interspersed with deep purple spots of an apoplectic character.

the tissues are more friable and lacerable even in the first stages, and in the progress of the disease we fail to find that evidence of plastic inflammation which accompanies ordinary sthenic pneumonia. Much yet remains to be done with regard to determining the actual morbific agent in many of the forms of disease with which we have to deal; and when we shall have arrived at a correct appreciation of the crases, which induce the palpable manifestations of inflammatory action, we may hope to reconcile theory and practice more fully than has yet been done; we may then also explain why such various modes of treatment are successful in combating symptoms, which, to our short-sighted vision, indicate the same disease, though in reality they are due to totally different causes. Thus, pneumonia, occurring in a system in which there is a predominance of lactic or lithic acid, would necessarily demand different remedies from one in which an alkaline or septic principle prevails. But to return to our legitimate sphere: the hypostatic form of pneumonia occurs in a variety of diseases, in which the patient is confined to the recumbent posture, and in which the system is much debilitated. Under the name of congestive pneumonia, it has been described by Mr. Erichsen as the most fertile source of the fatal issue of capital operations, after the patient has survived the first shock, and therefore calls for the special attention of the operating surgeon.

In this form, the congestion is by far the most prominent symptom; and though enough of vitality remains to give rise to some active symptoms, yet the debility of the patient, and of the organs of respiration, is so great as to prevent a reaction of a vigorous character. Mr. Erichsen[1] has analyzed sixty-two post mortems of individuals who died after operations, and has set down as pneumonic only those cases in which either one lung alone was affected, or else, in which some other palpable sign of inflammatory action was manifested in the cavity of the thorax, beyond a merely congested or softened condition of these organs, such as solidi-

[1] Medico-Chirurg. Transact. vol. xxvi. p. 29.

fication of their tissue, whether hepatization or splenification of it, the effusion of recent lymph or serum into the pleural sacs, or marked evidences of inflammation of the bronchial mucous membrane. The result of his analysis is as follows: There were twenty-eight in which there were evident signs of pneumonia; eleven in which the lungs presented the characters common to the first stage of pneumonia and passive congestion; nine where the lungs were diseased, but neither inflamed nor congested; and fourteen in which these organs were healthy, though many may have presented cadaveric congestion. A fact connected with this form of pneumonia, and pointed out by Mr. Erichsen, is, that while, in ordinary sthenic pneumonia, the right lung is most frequently affected, the left next, and lastly, both organs conjointly, here, the two lungs are most frequently affected together. In the twenty-eight cases in which pneumonia was traced, the right lung alone was affected three times, the left lung alone also three times; and both lungs together, though not to the same degree, in twenty-two cases.

In the pneumonia accompanying typhus, congestion is equally a predominant symptom, and requires the more to be carefully watched during life, as the insensibility of the patient and the general torpor of the nervous system allow it to run its course without producing (in many cases) any symptoms of inflammatory action beyond those obtained by the stethoscope. Cough, expectoration, and dyspnœa, may be absent, while we find extensive crepitation over a large extent of surface. In addition to the post-mortem appearances already detailed, we find the affected parts very soft, and the product is of an aplastic, jelly-like character, while there is no definite limitation to the disease. In some cases of atypic pneumonia, where we have reason to suspect an arrested secretion of bile in the liver, we find a genuine jaundice of other viscera, and then the frothy juice exuding from the cut surface of the pneumonic lung may present a yellow tinge, which has probably given rise to the term of bilious pneumonia. Again, in senile pneumonia,[1] the following varieties are described as belonging to the second stage; the lung, on incision, appears perfectly smooth and homogeneous, discharging a reddish, frothless serum, the inflammation occurring in patches, which are elastic, or soft; or, if granulations are present, they are much larger than in younger individuals. In the third stage, a peculiarity has been occasionally observed by Hourmann and Dechambre, which Hasse confirms; it consists in the purulent matter being sharply defined in spots of from one to two lines in diameter, which prove to be the irregularly dilated air-cells, often met with at intervals throughout the lungs of aged persons.

ABSCESS.

We have already seen that the formation of abscesses, such as we meet with in other organs and on the surface of the body, is rarely found in the lungs, notwithstanding the frequency with which inflammation attacks them. Yet a suppurative destruction of the tissue occa-

[1] See Hasse's Pathological Anatomy, p. 229.

sionally leads to this result, and we then find a cavity varying in size from that of a marble to that of an entire lobe, and presenting ragged parietes; it contains pulmonary debris and pus, which partially infiltrates the adjoining tissues, as there is no lining membrane; or the abscess may have discharged its contents, and we then only meet with the irregular excavations with jagged walls. Formerly, the frequency of pulmonary abscess was considered to be much greater than we now know it to be, because tubercular cavities were confounded with this lesion. Now that the nature of both affections is better understood, the error is less likely to arise; when we are in doubt, the history of the case, the situation of the abscess, the condition of the surrounding parts, the presence or absence of tubercular deposits in the lungs, are points that will aid us in establishing a correct opinion. Carswell has very justly remarked that the mere presence of pus must be carefully distinguished from suppuration. In the latter case, we have to deal with an active process, in which the tissues and bloodvessels of the part in which we find the pus are primarily engaged, or with suppurative inflammation; the process which John Hunter conclusively showed to be analogous to secretion. In the former, the pus is a foreign body in the blood, and may be conveyed to the seat of disease by the vascular channels from elsewhere, or be developed from the constituents of the vascular current within the vessels, and subsequently be discharged from them; a process more allied to chemical decomposition than to secretion.

METASTATIC ABSCESSES.

The mere presence of pus in the lungs, simulating abscesses, and, from the cause to which they are attributed, termed metastatic abscesses, or purulent deposits, constitutes a pathological phenomenon, which occurs more frequently in the lungs than the other parenchymatous viscera. This is quite in accordance with the known relation existing between phlebitis and the formation of secondary deposits; the lungs, as the receptacles of the entire mass of the venous blood, might therefore be expected to exhibit a peculiar liability to any morbid affection connected with this portion of the circulating system, a point of great importance in reference to the subject immediately under our consideration, as well as to other pulmonary diseases. The appearances by which we recognize these deposits are, spots of yellow pus, varying in size from a pin's head to a walnut, generally situated near the surface of the organ, and surrounded by a defined patch of deeply-congested tissue, which may present a color approaching to black; beyond this, the parenchyma is in a healthy condition, or, at all events, in a state totally distinct from the circumscribed disease. We generally find several of these abscesses in various parts of the lungs. The pulmonary tissue of the seat of the abscess may have entirely disappeared, or we may be yet able to squeeze out the pus, so as to show the normal structure.

Secondary abscesses in the lungs are almost invariably accompanied by similar deposits in one or several other viscera, and more especially

in that one which, as a depurator of the venous blood, is second in importance only to the lungs—the liver.

The universal concomitant of these abscesses is now well ascertained to be the suppuration of some distant part or organ, which induces phlebitis at the seat of primary injury. This may be the uterus after delivery, periostitis of the leg, fistula in ano, psoas abscess, or any other similar affection. The system must be in an ataxic condition to permit of the poisoning of the circulating current, and, though the introduction of pus into the blood is proved to be necessarily fatal, it is probable that the local injury would not give rise to phlebitis, unless a peculiar predisposition to it existed. Another question is, whether, after the occurrence of local phlebitis, the pus is necessarily conveyed from the part to each point that afterwards shows a secondary abscess, or whether a catalytic effect is produced on the blood, in consequence of what the older authors called a purulent diathesis. It certainly does not appear that the secondary local effects are a necessary consequence of purulent infection; but that this may prove fatal without producing them. M. Lebert gives two instances in which he noted the absence of metastatic abscesses, notwithstanding manifest purulent infection.

FIBRINOUS DEPOSITS.

We occasionally meet with deposits of a different character in the lungs, which Rokitansky appears to regard as identical with the purulent deposits just considered; but which certainly present different characters, and are connected with a different state of the system, unless, indeed, we adopt the view that pus and fibrin are essentially the same. These deposits occur in a more or less wedge-shaped form, with the base towards the pleura, near the surface of the lung. They present a light red or yellowish white color, surrounded by somewhat congested crepitant tissue, with a defined margin. The lung tissue is in a state of disruption, and the microscope exhibits the pulmonary debris in a granular stroma, in which granulated nuclei, varying much in size and shape, and some highly refracting globules of oil, are seen. The color, the consistency, the appearance of the tissue immediately surrounding these fibrinous deposits, as well as their microscopic characters, establish so many points of difference between them and purulent deposits, that we are scarcely justified in classing them together, especially as we do not observe that the two morbid products occur together in the same individual. Nothing can be more valuable than a simplification of the elements of disease, and the demonstration of the real identity of apparently different forms; at the same time nothing is more liable to lead us from the path of legitimate induction than hasty generalization. With regard to the origin of these deposits, we are inclined to regard them as an exudation from the capillary circulation, rather than as a result of fibrin being detached at distant points of the circulation, and carried by the current into the pulmonary parenchyma.

GANGRENE.

The most marked form which septic disease assumes in the lungs is presented in cases of gangrene; it is, as Dr. Watson observes, very seldom the result of acute inflammation, and is almost as uncommon as the formation of true abscess. It appears to be due to a peculiar constitution of the blood, or to a specific poison. Dr. Stokes has published some cases in all of which the patients had been habitual drunkards; the abuse of spirituous liquors does not, however, seem to exert a uniform influence in its production; for, in chronic alcoholism, as described by Dr. Huss,[1] this lesion has not been met with. It is also remarkable that ergotism, which has a peculiar tendency to induce superficial gangrene, is not accompanied by pulmonary sphacelus. The same applies to the analogous disease spedålskhed,[2] or the Norwegian leprosy. It presents two forms, the diffuse and the circumscribed. In the former, the lung tissue that is involved is broken up into shreds, which hang into a cavity filled with a fetid, purilaginous, discolored sanies, and through which the bronchi and vessels may yet be traced entire. The gangrenous portion presents a variety of hues, in the different shades of green, brown, and black. The surrounding parenchyma is infiltrated with ill-conditioned pus. An entire lobe, or even the greater portion of one lung, is found to be in this condition; though the upper lobe appears to be most prone to the affection. In circumscribed gangrene, which is more frequent than the former, we find one or more patches, varying in size and of irregular form, scattered through the lungs. There is much less tendency to involve the adjacent parts, and the course of the affection is more chronic than the former. Laennec describes the color of the mortified portion as black, with a greenish tinge—the texture as moister and more compact than that of the lung, and its aspect closely corresponding to an eschar, produced upon the cutaneous surface by nitrate of silver. The neighboring pulmonary tissue is in a state of inflammatory congestion; and, after the sphacelated spot has become detached, a false membrane, of a grayish, dirty-yellow color is formed, which secretes an ill-conditioned pus. At times, the membrane is formed even before the entire separation has been effected. If the gangrene involves the pleura, rupture and discharge into the pleural sac may ensue, or otherwise the bronchi may be the channels by which the evacuation is effected.

CHRONIC PNEUMONIA.

The existence of chronic pneumonia has been disputed by some authors; but both at the bedside, as well as in the dead-house, we find cases to which no other name can be given, though, in the present state of our knowledge, it is often difficult to define the exact limits of the acute and chronic forms. Another difficulty which has yet to be removed, in regard to this subject is, to determine whether chronic pneu-

[1] See Dr. Huss's Work on the subject; Alcoholismus Chronicus, &c., Stockholm, 1849.
[2] Dr. Daniellsen and Boeck, Om Spedålskhed, &c. Christiania, 1847.

monia is essentially different from the intercurrent pneumonia associated with tubercular phthisis, and if so, whether there are any marked characters by which in the dead body, where we only have to consider it at present, it can be recognized. Andral, who looks upon it as a frequent idiopathic affection of the upper lobes, and as that form of pneumonia which, from this circumstance, has not met with the proper attention, differs materially from the views promulgated by Hasse, and those who look upon chronic inflammation of the lungs as essentially connected with the tubercular cachexia.

The chief feature in chronic pneumonia, which we find equally dwelt upon by almost all observers, is the hypertrophy of the intervesicular tissues, owing, probably, to a deposit of an albuminous character in them. Rokitansky, in contradistinction to his croupous form of pneumonia, gives the name of interstitial pneumonia to a second form, which he regards as identical with the chronic form of other pathologists, as, for instance, Andral and Hasse; the difference is rather one of names than of reality, for Rokitansky also admits that the walls of the air-cells are often implicated. He describes the inflammation as commencing between the pulmonary lobules and the smaller groups of air-cells; the tissue, unless much carbonaceous matter be present, becoming of a pale red color, and being swollen by albuminous infiltration, while the air-cells are either pale or more or less compressed, in proportion to the swelling; or if they are involved in the inflammation, reddened, and sometimes finely granular. In the progress of time, the interstitial infiltration becomes organized, coalesces with and obliterates the air-cells, and finally converts them into a similar tissue. In some cases this process may terminate in suppuration; in others it extends from lobule to lobule, at the apices of the lungs, and is frequently combined with circumscribed pleurisy.

Dr. Williams describes a chronic form of pneumonia, in which the hepatized portion, owing to the thickening of individual vesicles, assumes an oolitic aspect. He is of opinion that consumption may originate in this species of pneumonia without the pre-existence of any distinct tuberculous disease. It is not impossible that some of the fibroid contractions of the pulmonary tissue, which we meet with, particularly at the apices of the lungs, may be due to an arrest of chronic pneumonia as well as to previous pleuritic inflammation; we occasionally meet with depressions in otherwise healthy lungs, which are unconnected with emphysema, and for which no other explanation can be offered than a foregone inflammatory condition of the interstitial tissues of an aplastic character, an opinion in which we are also borne out by the authority of Dr. Williams.[1]

[1] Principles of Medicine, p. 313. London, 1843.

CHAPTER XXX.

ADVENTITIOUS PRODUCTS IN THE LUNGS.

We now arrive at the consideration of a subject which, both in its scientific and in its practical bearings, is one of the utmost importance in connection with the morbid anatomy of the lungs more than with that of any other organ, the deposit of tubercular matter. In the section devoted to the general pathology, we have entered into the prevailing views entertained with regard to the nature of tubercle. The affinity between lymph and tubercle insisted upon by older writers, and recently more fully urged by Dr. Alison, Dr. C. J. B. Williams, and, under the appellation of fibrinous crases by Rokitansky, must not be lost sight of, although it only states the one fact, that tubercle is a secretion of a morbid constituent of the blood, in different terms, without actually defining the character of the change. It leaves the relation between the salts of the circulating fluid and the albuminous constituents untouched, nor does it determine whether any chemical agent which the exuded matter may meet with, in any way alters its nature, and thus converts what, in the first instance, may have been innocuous, into a deleterious agent. We are led to make these remarks by the interesting experiments lately published by Dr. Parkes,[1] on the "Precipitation of Albumen by Acids and Neutral Salts," which promise to throw much light on the ultimate constitution of tubercle, as well as upon many points connected with its origin and metamorphoses. Our limits will not allow us to detail the processes employed, but the conclusions arrived at are sufficiently important to justify their introduction at this place; they are: 1. That the albumen in the serum of the blood is occasionally in a condition in which it is not precipitated by an acid and a neutral salt. 2. That if that albumen be exposed for some time to a strong alkaline solution it ceases to be precipitable by acids and salt. 3. The continued action of acetic acid on serum also annuls the action of acid and salt on albumen; hence it is evident that the albumen, as it exists in the serum, is usually in that condition in which it is most easily precipitated by acids and chloride of sodium; but that it very readily passes out of this condition under the continued influence of acids and alkalies, and is then, in all probability, incapable of reassuming it. Dr. Parkes also shows that, under certain circumstances, the albumen is found in a corpuscular condition, viz: in the form of round globules and cells of different sizes, sometimes inclosing each other, and forming conglome-

[1] Medical Times, 1850, July 27, and 1852, July 3.

rates, and sometimes completely resembling the eye-globules of fat. The former observations indicate the nature of the bio-chemical changes, which obtain, when an aplastic deposit, such as tubercle, is effected, while the latter assist in explaining the fact that we meet with what is called fat or oil, in varying quantities in all tubercular deposits, and also aid in the intelligence of that morbid state of nutrition known under the term of fatty degeneration.

The lungs are the organs which, above all others, are liable to tubercular deposit; a circumstance easily explained, if we admit that tubercle is derived directly from the blood, as the lungs are the chief organs of hæmatosis, and anything tending to check the purification and arrest the circulation of the current would also induce an elimination of the morbid element. Hence, too, the apices, from being inclosed in a less yielding case, mechanically favor the effusion more than the lower lobes. We are unable to reconcile with this fact the categorical statement of Rokitansky, that the main feature of the tuberculous crasis or diathesis consists in arteriality or a predominant arterial development of fibrin, since, if this were the case, we should certainly expect to find the tuberculous deposit more frequently in the brain and kidneys than in the lungs, and more commonly in the interstitial tissue of the latter, as a secretion from the bronchial arteries, than in the respiratory vesicles. That the deposit is extravascular, is undoubted. Messrs. Gulliver and Addison have shown that it is effected on the surface of the air-cells, or under the basement-membrane, though our own observations lead us to believe that the primary deposit is invariably at the point of smallest resistance; and hence, in the first instance, always into the free cavities. That, when these are filled, the morbid plasma may be deposited in the interstitial parenchyma, is certain; Dr. Williams is even of opinion that tuberculous matter may form within the bloodvessels themselves, as he has repeatedly found something presenting all the external characters of yellow tubercle in the bloodvessels of tuberculous lungs. "In fact," he continues, "wherever fibrin may coagulate, there its degraded form, tubercle, may occur. It is but in accordance with the general rules of morbid formations, that the deposit of tubercle should undergo modifications both in form and chemical constitution, according to the organ in which it appears and the habit of the individual; hence, though its characters are sufficiently uniform to justify our treating it as a disease *sui generis*, numerous variations present themselves, owing to the influence to which we have alluded, which have given rise to conflicting statements. The microscope has more especially appeared to complicate, instead of solving the difficulties, owing to the presence of epithelium-corpuscles, nuclei, and proper corpuscles (as in the glands), in most of the parts where tubercular deposit occurs. The greater or less congestion, irritation, and inflammation coincident with tubercular deposit, has no less confused the doctrines relating to the subject; though, if we take the whole complex of pneumonia, and analyze the successive stages which we meet with, we shall find that the real obscurity is not so great as it at first sight appears. A material difficulty in the examination of diseased lungs is that, in all the early stages, the quantity of air contained in the tissue is so great as to constitute a considerable impediment to the

minute investigation; and, though the examination of dried or seasoned specimens may assist in elucidating some points of morbid anatomy, it is manifest that the most important questions will remain unsettled, unless we arrive at our conclusions from what we discover in the most recent examples.

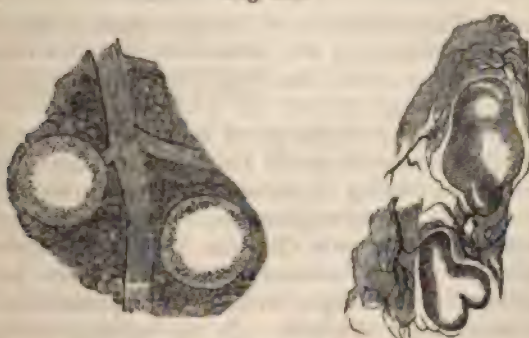
The primary deposit of tubercle takes place, in a semifluid form, into the vesicular cavity, which it distends so as to form a round point, of the size of a small pin's head, visible to the naked eye. It forms a

Fig. 188.

Miliary tubercle, scattered throughout the pulmonary tissue, forming translucent, grayish, and circular points, of the size of pins' heads.

translucent mass, which entirely blocks up the cavity, stopping abruptly at the entrance of the ultimate bronchule, like a bullet that exactly fits its mould. The fine basement-membrane of the vesicle is more or less obscured by the dark ring of exudation-corpuscles covering it, and which

Fig. 189.



Microscopic appearance of miliary tubercle. The drawing to the left shows the site of the deposit and its relation to the pulmonary tissues; the effusion has taken place into the air-vesicle, which is distended into a globular ball. The drawing to the right exhibits a section of miliary tubercle without the bronchules, surrounded by congested vessels.

we invariably find wherever there is any irritation accompanying the deposit, whether of a primary or a consecutive character. The ultimate divisions of the bronchi escape the power of unassisted vision, but, in a successful section, may be clearly seen with a power of twenty diameters.

This deposit scarcely encroaches upon the surrounding parenchyma; but when, instead of solitary vesicles being the seat of the exudation, whole clusters of breathing-cells are at once charged with tubercular matter, the microscopic appearances are rather those of a mould of grapes taken in blanc-mange, and we no longer detect the interstitial spaces, partly from the increased opacity of the deposit rendering it impossible to transmit light through a section of any thickness, partly from the compression exerted upon the interstitial tissue obliterating it. The obliteration of the pulmonary tissues progressively increases; the rounded form of the vesicular structures is gradually destroyed; by mutual pressure they assume an hexagonal or polygonal shape, a mere line indicating the points of separation; the bronchules leading to this morbid mass are seen to be obliterated, dwindling down, near the tubercular aggregation, to a point, and ending, like a ligatured artery, in a mere

Fig. 190.

Microscopic appearance of a mass of miliary tubercle, in close aggregation.

Fig. 191.

Hexagonal appearance caused by the mutual pressure of the air-cells, filled with yellow tubercular matter, with obliteration of the bronchules leading to the lobules. Magnified 60 diameters.

thread. The translucent character of the tubercle, by this time, is utterly lost; it has assumed, partly owing to original constitution, but more, in our opinion, owing to secondary changes which have taken place in it, an opaque cheesy appearance, of a creamy or clayey color, resembling fresh Stilton or pale Cheshire, both in hue and consistency.

If the tubercular matter be analyzed by the higher powers of the microscope in these various stages, we find that it presents corresponding differences in its ultimate elements. In the earliest and most recent form we see a fine molecular blastema of an almost homogeneous appearance, in which faintly, but definitely, the outline of lighter particles of an oval or circular shape, and from 0.0003″ to 0.0002″ in diameter, may be traced, themselves less granular than the stroma in which they are imbedded. The tubercle-corpuscle contains no nucleus, a point which, as M. Lebert has shown, is confirmed by the addition of acetic acid failing to bring it out, and which serves to distinguish it from similar formations. The tubercular elements are not, as has been asserted, a

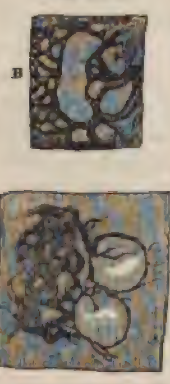
mere modification of epithelium; but though we frequently find epithelium mixed up with, and possibly, at times, closely resembling the more developed forms seen in tubercle, they possess a character and evolution of their own. The denser the deposit, the more uniform the conversion of the blastema into definite oval, or somewhat angular corpuscles, with a definite cell-wall and granular contents. The corpuscle is not a mere lamina, but appears to have an ovoid shape. A new process of disintegration now appears to ensue, and we see the elimination of a large number of highly refracting particles, hitherto considered as fat or oil, but possibly, according to the experiments of Dr. Parkes above alluded to, peculiar modifications of albumen. When the tubercular deposit has arrived at this stage it appears that an arrest of the morbid process may take place, it may become obsolete, and undergo changes that enable the system to bear its presence without serious inconvenience, or the morbid process advances with more or less activity, inducing a softening of the deposit, and a destruction of the pulmonary tissue, all of which we shall have occasion to investigate further on.

We now turn to consider the morbid phenomena and changes in the pulmonary tissues associated with the deposit of tubercle. These are of a more or less active character, according to the exciting and predisposing causes operating in the individual case, and according to the general habit of the patient. It has, therefore, become customary to form two groups of pulmonic tubercular diseases, the acute and the chronic. Other authors have preferred dividing the subject, according to the characters presented by the tubercular deposit, into the gray or yellow varieties, or, as Rokitansky more particularly has done, according to the place of deposition, into the interstitial and infiltrated form; the former term being applied to tubercular matter exuded into the intervesicular textures, the latter to its occurrence within the cavity of the vesicles. We have already seen that the density of tubercle varies with the period of its deposit, and with the amount of pressure it may be accidentally exposed to. No important characteristic seems to depend either upon its color, except that the most decidedly gray and translucent form is the more recent, and most commonly connected with a more actively inflammatory condition of the parts; the same influences which induce the change of form and consistency undoubtedly modify, together with the molecular alterations that coexist, the color of the deposit. Still, the deposit may, from the first, present an opaque yellowish appearance, and, as Carswell observes, the gray, semi-transparent substance does not necessarily precede the formation of the pale yellow or opaque tuberculous matter. After the tubercular matter has become firm, it may, at some future period, be converted into a pulpy grumous fluid, presenting various colors of a greenish, red, brownish, or other hue, dependent upon the admixture of serum, blood, or pus, which pervade the substance of the tubercular matter, loosening and detaching it. From this it follows that there must be some error in the commonly received doctrine, that the softening always commenced in the centre of the deposit. We commonly find a depression in the middle of an accumulation of tubercular matter. This has been attributed by Laennec to softening. Carswell's explanation is as follows: "When tuberculous

matter is found in the lungs it is generally contained in the air-cells and bronchi. If, therefore, this morbid product is confined to the surface of either, or has accumulated to such a degree as to leave only a limited central portion of their cavities unoccupied, it is obvious that when they are divided transversely the following appearances will be observed. 1. A bronchial tube will resemble a tubercle having a central depression, or soft central point, because of the centre of the tube not being, nor ever having been, occupied by tuberculous matter, and because of its containing a small quantity of mucus or other secreted fluids. 2. The air-cells will exhibit a number of similar appearances or rings of tubercular matter, grouped together, and containing in their centre a quantity of similar fluids. When the bronchi or air-cells are completely filled, the tuberculous matter presents no such appearances as I have described, and hence the reason why tubercle, in such circumstances, has been said to be still in a state of crudity, or that condition which precedes the softening process. Softening begins most frequently at the circumference of the tuberculous matter, or where its presence as a foreign body is most felt by the surrounding tissues. Hence, the reason why softening is frequently seen making its appearance in several points of an agglomerated mass of this substance, which has included within it portions of the tissues in which it was formed. This is frequently observed in the lungs, and cellular tissue in other parts; whereas, in the brain, the substance of which has, from the commencement, been separated and pushed outwards by the tuberculous matter, the softening process begins, and is always most marked on the circumference of this morbid product."

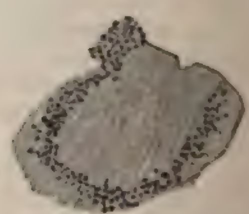
As we have assumed that there is essentially but one form of tubercle possessed of characters sufficiently definite to justify the classing under one head varieties which owe their differences to accidental or trifling

Fig. 192.



Microscopic appearance of minute vessels, surrounding air-vesicles in tubercular pneumonia, and miliary tubercle.

Fig. 193.



A section of an air-vesicle filled with yellow tubercle, and surrounded by exudation-corpuscles.

circumstances, and as we believe that the diathesis of the blood and the general constitution of the individual are essentially the same, in which the deposit is effected, so also are the symptoms and local changes

accompanying the elimination of tubercle more or less the same in the different cases. They are essentially those of a low type of inflammation. The primary deposit of tubercle is always accompanied by an increased afflux of blood to the part; hence the great danger in scrofulous subjects of the slightest catarrhal inflammation, because the diathesis being present, nothing is required to induce the deposit but a congested state of the parts generally liable to it. The further evidence of an inflammatory process is given in the constant occurrence of exudation-corpuscles in the immediate vicinity of the air-cell into which the effusion is taking place, or has recently been effected. These corpuscles, whether in the form of cells with a proper wall, or of mere aggregation-molecules, are seen entirely coating the air-vesicle if we obtain a view *à posteriori;* or, if we succeed in making a transverse section, a dark ring formed by these corpuscles, and probably external to the basement-membrane, between the vessels and vesicular coat, is seen bounding the tubercular matter. This point the more deserves attention, because in certain experiments made by Gluge to disprove the views entertained by Dr. Addison, that the morbid state of the lungs accompanying acute miliary tubercle is of an inflammatory character, stress has been laid upon these corpuscles being distinct from tubercular matter, and that the error had arisen from their being in the previous experiments mistaken for the cacoplastic exudation itself. Their characters are certainly well defined, and it is this point that, in our opinion, confirms the views of the inflammatory nature of the process, which, not only in miliary, but in every form of tubercular deposit, accompanies its elimination. In the words of Dr. Graves, the most important thing for the student to impress on his mind, with regard to all cases of phthisis, is, that the pectoral symptoms, of whatsoever nature they may be, are caused by scrofulous inflammation. The hæmoptysis which is found to precede or accompany two-thirds of all cases of pulmonary consumption, and is so constantly connected with tubercle, that Louis has laid it down as denoting with infinite probability the actual presence of some tubercles in the lungs, at once furnishes a proof of the manifest congestion accompanying the lesion, and of the peculiar aplastic condition of the blood, by which, under comparatively slight exciting causes, it is forced through the coats of the vessels. This hæmoptysis also confirms the view we have advocated with regard to the intra-vesicular character of tubercular effusion; we never find any symptoms of apoplectic hemorrhage, or hemorrhage within the pulmonary parenchyma accompanying tubercular disease, but it is invariably an exhalation on the free surface of the respiratory membrane. Now it is fair to conclude that if the tubercular blastema were effused interstitially in the first instance, we should here also, in a proportional number of cases, find the results of hemorrhagic effusion, which is not the case.

With reference to the mode in which tubercle is distributed through the lungs, we find that it presents three varieties, which are generally tolerably defined in the lungs of one individual, though occasionally the several forms of deposit occur together. Hasse describes them in the following terms: "First, they are found single, isolated, and more or less uniformly disseminated miliary tubercles; secondly, they are found

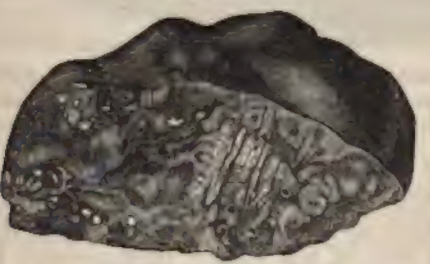
in scattered groups, assuming various forms, the tubercles being now loosely collected together, now closely connected either in a regular mulberry shape, or in clusters of indefinite form, aggregate tubercles; thirdly and lastly, they are found densely crowded throughout a portion if not the whole of a lobe, so as to constitute seemingly but one coherent mass, tuberculous infiltration." We also coincide with the observations which follow the preceding extract: "Their mode of distribution is naturally influenced by their mode of development. Where they form rapidly they are the more equally dispersed, where slowly they become in the same measure subject to the law which causes them to accumulate in the summit of the lung, and from thence downwards gradually to decrease in compactness, with a proportionate tendency to run into groups. The aspect of the individual tubercles is also modified by the manner of their distribution, the gray variety in particular becoming whiter and losing its transparency, when densely congregated."

A species of antithesis exists in the lungs between the tendency to tubercular deposit in their apices and the liability of the lower lobes to idiopathic inflammation. This may be accounted for by the encouragement to deposition from the blood offered in the apices of the lungs, by the lesser expansibility of these parts. The movement of the clavicle and two upper ribs is very small compared with that of the lower ribs, and the intercostal muscles are more rigid here than below, circumstances that are enhanced by a sluggish habit, while they favor any accidental impediment to the aeration of the blood. This, in its turn, diminishes the rapidity of the current as well as its quality; hence the morbid deposition ensues, and, as the cavities of the air-vesicles are the points of least resistance, the effusion is effected into them. We have already mentioned that the more rapid the course of phthisis, the more generally we find the deposit diffused through the lung. In a case of acute miliary tubercle, from which one of our illustrations is taken, the bases and the apices of both lungs were uniformly affected; both organs being studded throughout with the deposit, which had probably taken place but a few days previous to death, in a subject debilitated by rheumatic affection of the heart and central softening of the brain. Both lungs, according to the law of symmetry, present a proclivity to tubercular disease; Laennec considered that a greater tendency existed on the left than on the right side, owing to his having met with five instances in which it was limited to the left lung, while only two occurred to him in which the right alone was the seat of disease. Other observers have failed to confirm Laennec's observation, and have asserted either that the right lung was more frequently affected, or that no difference was traceable. If a difference exists in the early stages of the disease; and it is probable that, in its progress, the balance is restored; our own experience decidedly tends to show a predominance on the right side, in the incipient period.

The questions which next suggest themselves for our consideration are the ulterior processes which the tubercular matter and the pulmonary parenchyma undergo after the deposit has been effected. They may be of an opposite character; either exhibiting a progressive development, a tendency to destruction of the part and dissolution of life;

or manifesting a retrograde movement, with evidence of the product of the disease becoming obsolete, and the morbid germ being extinguished. After the first deposit of tubercle is completed, the morbid diathesis may, as it were, have exhausted itself, and the process enters into a state of abeyance, all active symptoms being arrested; or the inflammatory action is perpetuated and the course is continuous to the fatal termination. In the former case, every recurrence of bronchitic or pneumonic inflammation finds a nidus of tubercle around which fresh matter is deposited, or which becomes an additional source of irritation. In both cases a process of fusion takes place, serum and pus are secreted from the parts in which the morbid deposit has been effected, the intervesicular septa are more and more absorbed and broken down, the softened matter finds its way into the larger bronchi and is expectorated, a mixture of pus, mucus, melanotic, oily, and granular matter, interspersed

Fig. 194.



The apex of a lung affected with tubercular pneumonia; the limitation of the disease was defined by a sharp line bounding the inflamed tissue, which surrounded the tubercular deposit.

with epithelium and the elastic fibres of the lungs, and the result is a cavity in the pulmonary tissue. The walls of this cavity may be more or less rugged, and be more or less lined with tubercular matter, or present no traces of it, according to the date of its formation; these tubercular cavities were formerly often mistaken for genuine abscesses —we find them in all numbers; there may be but one, or so many as to give the entire lung a riddled or honeycomb appearance when cut into; they vary equally in size, from a pea to a man's fist and more. The communication between the abscess and the bronchus passing out of it, resembles a fistulous opening, the peculiar relation of which to respiration causes many of the phenomena of auscultation. It is rare, as Laennec observes, to find a single cavity; the excavation is commonly surrounded by crude and miliary tubercles which gradually soften, are then discharged into the main cavity, giving rise to the anfractuosities which we commonly observe. The excavations are often traversed by bands of pulmonary tissue infiltrated with tubercular matter, and compared by Laennec to the columnæ carneæ of the heart. He also suggests that they were mistaken by Bayle for vessels passing across the cavities, for vessels are scarcely ever seen in this position. The progress of the tubercular deposits causes the obliteration of the vascular channels as of the other parts of the normal tissue, and we only find the vessels of a larger caliber in the vicinity of the cavities. The

bronchules, and successively the larger air-tubes, are subjected to the same destructive agency, until the power of resistance caused by the

Fig. 195.

A lung, exhibiting extensive tubercular disorganization throughout its upper lobe, which is almost converted into one rugged cavity. The pleura is very much thickened; intimate adhesion has taken place between the upper and lower lobe, and the tubercular deposit is seen encroaching upon the latter.

stronger walls of the bronchi of the first and second order is too great to be overcome by the morbid process.

Instead of a mere uneven rugged surface, as if the lung had been mouse-eaten, the walls of the abscess often present a uniform velvety appearance, and are invested by a false membrane, which may assume a considerable thickness of one-third or half a line. At the earlier

Fig. 196.

The apex of a lung containing numerous cavities, with tubercular deposit intervening. The large cavity, and several of the smaller ones, are lined with an adventitious membrane.

periods of disease, the membrane is of slight consistence and easily separable. The membrane is described by Hasse as being formed out of the hepatized crust that encircles the enlarging cavities, and which,

instead of becoming softened and liquefied through purulent formation, merely becomes saturated with coagulable substances more akin to the organism; "but," he continues, "no sooner is the tubercular diathesis revived and aggravated by a catarrhal or inflammatory attack, than the protecting false membrane liquefies, and purulent secretion, mingled with tubercle, again sets in. The cavity itself, which under the above circumstances seemed to shrivel and contract, gains size, new vomicæ form, the lung becomes more and more deeply involved, and the mischief terminates only in death." The tissue beyond the lining membrane may be in a healthy condition, or present tubercular deposit in various degrees, but generally containing much melanotic carbonaceous matter. In the majority of instances both lungs are found to present excavations. Louis states that in one-sixth only of his total cases of phthisis, they were limited to one or the other lung, and when present on both sides were of different size; in somewhat less than one-tenth of his cases, both lungs were the seat of enormous excavations, equally large on both sides, and in another tenth, where the cavities presented but small or moderate dimension, these dimensions were the same in both organs. Large cavities (of the size of a goose's egg or a clenched fist) Louis found to occur in about one-half of the cases, and with equal frequency in each lung; in the remainder of his subjects he found cavities of the size of an ordinary-sized apple or walnut.

The complications of disease occurring in the course of tubercular phthisis and at different periods, are inflammations of the mucous membranes of the air-passages of the pulmonary parenchyma and of the pleura. Of the appearances presented by the first, and of the relative frequency of occurrence in them of ulcerations in consumption, we have already spoken. There can be no doubt that the acrid character of the expectorated matters very much favors the ulcerative process. The frequency with which slight attacks of pneumonia supervene in the course of phthisis, is not surprising, if we look upon it merely as an exacerbation of the process actually constituting the disease—it is rather in each case the cause of an extension of the morbid deposit, than the result of the previous elimination of tubercle. The pleurisy accompanying tubercle, may supervene at various periods, and by the adhesions it causes and the consequent increased immobility of the lung, it necessarily much favors the development of the tubercular process. That the tubercular deposit has a tendency to excite pleurisy, is evident from the frequency with which the latter occurs at the apices of the lungs, often forming a complete cartilaginoid cap from which it is difficult or impossible to detach the lung entire. When tubercular excavations approach close to the pleural surface, and are not preceded by thickening of the pleura, as generally occurs, a perforation may take place, inducing effusion of air and liquid into the cavity and secondary inflammation; we then have to deal with hydro-pneumothorax, upon which a rapidly fatal issue is almost certain to follow. Mere cellular adhesions, according to Rokitansky, cannot prevent this termination; they are, in part, mechanically loosened by the effusion from the cavern, and being involved in the pleuritic process, they are in part likewise destroyed in the exudation. Rokitansky describes three forms in which the communication between

the tuberculous cavity and the sac of the pleura may be established; the pulmonary pleura may be detached from the affected surface by the mere force of the air rushing in so as to form a bulla, which afterwards bursts; it may be converted into a whitish eschar which tears or becomes detached unbroken, or the pleura, together with the infiltrated parenchyma surrounding the cavern, may become gangrenous and be converted into a purilage. The complication of pulmonary tubercle with tubercular deposits in other organs is of very common occurrence; Louis[1] found tuberculous ulceration in the intestines in five-sixths of the cases he examined, the lymphatic glands were affected in the following order of frequency, the bronchial most, next the mesenteric, the axillary, mesocolic, lumbar, and cervical; the spleen and kidneys exhibited tuberculous deposit in about one-sixth of the cases; several times the prostate was found more or less transformed into tuberculous matter, and tubercular deposit was also met with in the cerebral arachnoid, though the frequency is not stated. The prevailing condition of the liver in pulmonary phthisis is one of fatty degeneration. Louis found it fatty in one-third of his cases. With regard to the coexistence of pulmonary tubercle, with tubercular deposit in other organs, the general law has been established, that wherever, after the age of fifteen, tubercles present themselves in any organ of the body, we are certain also to meet with them in the lungs.

When the tubercular deposit in the lungs does not advance and undergo the progressive changes which we have described, a process of obsolescence occurs which appears to consist in certain chemical changes in the tubercular matter, accompanied by an extinction of the peculiar crasis which had given rise to its elimination, and followed by certain secondary alterations in the surrounding tissues by which they are adapted to the requirements of the case. These we find to exhibit two distinct forms, which probably depend upon a difference not yet properly appreciated or well defined, in the diathesis primarily giving rise to the deposit. They are characterized by a fibrinous or a calcareous metamorphosis. In the former case we find the tubercle assuming a more dense and leathery character, of a semi-cartilaginous consistency, drying or shrivelling up as it were, accompanied by a contraction of the superimposed tissue. If we examine the yellowish deposit under the microscope, we find mixed up with the ordinary forms of tubercle a distinct fibrinous formation exhibiting a linear, striated appearance. In the latter, a conversion of the tubercular matter seems to be effected into a chalky substance, at first moist and soft, gradually, owing to absorption of the fluid constituents, becoming harder and drier, and, at the same time, shrinking from its previous dimensions; thus, at times, we find, to use Hasse's words, "that a considerable portion of the lung, as may be inferred from the size of the bronchial tubes leading thither, becomes reduced to a hard shell, holding in its centre a chalky tubercle no bigger than a pea." The chemical characters of these formations have already been alluded to; but it may be well to remind the reader that, although termed chalk, they do not consist of carbonate of lime,

[1] Researches on Phthisis, Syd. Soc. Ed. p. 150.

or of the elements of gouty chalkstones, urates of soda, but that they are mainly composed of chloride of sodium and sulphate of soda, as demonstrated by the researches of M. Boudet.[1] Lebert has also repeatedly observed cholesterin in chalky tubercles. The black pigment, which is at times met with to a considerable amount in tuberculized lungs, and still more in the bronchial glands, does not present any different features from that commonly found in the pulmonary parenchyma. It appears to consist of pure carbon, and may present a mere amorphous granular form, imbedded in and scattered irregularly through the tissue, or it is found inclosed in an epithelial cell. That it bears some relation to the defective oxygenation of the blood, is manifest from the normal tendency to the deposits being greater in proportion to the advance of life; and it has appeared to us to accumulate very rapidly in some cases of chronic inflammation. The presence of this black matter in the expectoration of phthisical patients in part gives rise to the grayish or dirty tinge we frequently observe.

Obsolete tubercle is surrounded by a dense capsule resulting from inflammatory induration. It is commonly found at the apices of the lungs, and may be easily felt on handling the part, and, if near the surface, their effect is rendered visible by a drawing in and puckering of the pleural surface.

The metamorphosis of tubercle into fibrinous or cretified masses must be regarded as evidence of a curative tendency. Whether we are justified in assuming, as it appears Carswell[2] does, that an entire ab sorption of tubercular matter may take place without a metamorphosis of this kind, or without the formation of a cavity, is a question which

Fig. 197.

Cicatrix at the apex of a lung, resulting from the previous arrest of tubercular disease.

we are not prepared to answer positively, though there is no certain proof to the contrary. That the healing process is not limited to the first stages of the disease, but is also seen after the formation of cavities, is established by the unanimous testimony of the best observers. Andral remarks that traces of cicatrization are found in individuals who, at one period of their life, have been subject to a severe affection of the respiratory organs, which was regarded as phthisis, or in such as have been cured of a previous pulmonary attack, but have suc-

[1] Recherches sur la Guérison Naturelle ou Spontanée de la Phthisie Pulmonaire. Paris, 1843.

[2] Elementary Forms of Disease, Art. Tubercle.

cumbed to a subsequent one of the same character; or, lastly, in persons who from the first day of their cough have continuously grown worse, in whom, therefore, after the cicatrization of one cavity new ones had formed. The cavities may disappear altogether, leaving a dense white fibrous tissue, ramifying irregularly in the surrounding tissue; or the obliteration is incomplete, the cavity remains partially open, and the character of the lining membrane undergoes a change assimilating it to a serous membrane, or, which is more commonly the case, converting it into a vascular villous covering, resembling a mucous membrane. Rokitansky states that, in the latter case, aneurismal dilatation, or a gelatinous degeneration of the vessels subjacent to the membrane, is liable to give rise to hemorrhage into the cavity, which either proves fatal, or else, by coagulating and plugging up the vessel, becomes a further means of obliteration and ultimate cure.

Before quitting the subject of pulmonary tubercle, we have to allude to certain differences which exist between the manifestation of the disease in early life and later years. In the former instance, the lungs are much less liable to become the seat of the deposit than they are in the latter; it is more commonly simultaneously deposited in a greater number of organs; and while, according to the extensive researches of Messrs. Rilliet and Barthez, which are confirmed by those of Dr. West, the yellow form of tubercle largely predominates, there is a remarkable immunity from tubercular cavities. The cavities that do occur are much smaller in proportion, and, though occasionally very numerous, do not give rise to the same amount of destruction of the pulmonary tissue that we see in the adult. The differences alluded to may be accounted for, partly by the greater share the nutritive organs take in all the functions of early life, and partly by the proclivity existing in the lungs to lobular inflammation. The latter circumstance renders it probable that a minute examination of tubercle in the infant, would exhibit a greater production of pus coincidently with the elimination of tubercular matter, than is found in the adult. We extract the following table from Dr. West's classical work on the diseases of childhood, as the best illustration that can be offered of the relation borne by tubercle to the different organs of the body in early and advanced life.

Of 100 instances in which tubercle was deposited in some of the viscera, it was present in

	Children from 1 to 15 years. According to Rilliet & Barthez.	Adults from 20 years and upwards. According to Louis.	According to Lombard.
The lungs	84	100	100
Bronchial glands	79	28	9
Mesenteric do.	46	33	19
Small intestines	42	33	0
Spleen	40	13	6
Pleura	34	2	1
Peritoneum	27	0	0
Liver	22	0	1
Large intestines	19	10	0
Membranes of the brain	16	0	2
Kidneys	15	2	1
Brain	11	0.8	2
Stomach	6	0	0
Heart and pericardium	3	0	0

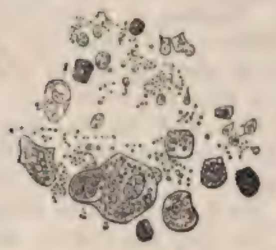
The table, as Dr. West remarks, shows not only that the liability of certain organs to become the seat of tubercle, is different in childhood from what it is in the adult; but, also, that tubercle is simultaneously deposited in a greater number of organs in the young than in the old.

CANCER.

Malignant disease of the lungs is not a common disease. Mr. Adams's research has, however, shown that it occurs more frequently, both as a primary and as a secondary affection, than was supposed to be the case by Bayle, who, in 900 subjects examined, only once met with cancerous growths in these organs. Both Laennec and Bayle only found the medullary variety; other forms have since been met with, but the encephaloid is that which vastly preponderates. Hasse has seen an instance of colloid cancer; in the Reports of the Pathological Society of London for 1849–50, a case of primary fungus hæmatodes of the lung is

Fig. 198.

Fig. 199.



Infiltrated cancer of the lung, with its microscopic elements. The lighter part is that containing the deposit; it was of a brownish-red tinge, and of greater density and hardness than the unaffected parts. It occurred in the right lung of a young woman, aged 34, whose right bronchus was surrounded with a mass of medullary cancer; the right kidney also contained a large growth of the same kind, and the 11th dorsal vertebra, especially its right side, was extensively destroyed by the same disease.

detailed by Mr. Adams. The only fact by which it appears that we may determine the primary or secondary character of the deposit, is the circumstance of the lungs either being the sole, or, at any rate, the

chief seat of the disease. It is stated by high authorities, that the former always assumes the infiltrated character, while the latter appears exclusively in nodules or isolated tumors scattered through the lungs. This distinction cannot, however, be rigidly maintained. The pulmonary texture entirely vanishes in the malignant growth, while the surrounding tissue is compressed and its functions interfered with. The neighboring lymphatic glands are invariably involved in the degenerative process. With regard to the parts of the body from which cancer spreads to the lungs, Hasse remarks "that the bones and testicles appear to furnish the most frequent starting-point; and numerous examples tend to show that surgical operations for the removal of cancer in those parts, are very speedily followed by its transition to internal organs. Many instances are adduced in which the skin and the mammary glands, the uterus, the liver, the membranes of the brain, were first assailed. I have seen a very remarkable instance consecutive to primary cancer of the submaxillary gland. On the other hand, cancer, in organs whose veins are tributary to the portal system, does not appear to spread to the lungs, although it is known to lead very often to corresponding disease of the liver." To this we would add that malignant disease, occurring in the mediastina, as it frequently does, does not appear to possess a great tendency to affect the lungs. We often see large masses of cancerous growth occupying these parts, and, perhaps, causing death, as well by suffocation as by exhaustion, without a trace of malignant disease in the lungs, although there is proximity of tissues as well as an intimate relation by the bloodvessels. The only case of the pancreatic variety, to use Abernethy's apt designation, that has lately fallen under our observation, entirely filled the upper part of the anterior mediastinum, and infiltrating the pectoral muscles of the right side without affecting the lungs, otherwise than by pressure. A good instance of reticular carcinoma of the posterior mediastinum was exhibited by Dr. Jenner, at the Pathological Society of London,[1] which, however, slightly encroached upon the root of the lung; we had an opportunity of examining it, and could, therefore, confirm the fact of the reticular character. Neither of these varieties has been seen to occupy the pulmonary texture. Whether the peculiar functions of the lung influence the nature of the deposit, or whether this depends upon some other cause, there is no evidence to show.

A law universally adopted, and one that appears perfectly consistent with the inherent tendency to endogenous multiplication in cancer, and the absence of this character in tubercle, is, that the cancer does not coexist with tubercle. We do not wish to assert that there are no exceptions from the rule; but they are so rare, and in those instances on record the diagnosis generally admits a reasonable doubt, so that the law is in no way invalidated.

In connection with cancer, we have to allude again to an excessive deposit of carbonaceous matter in the lungs; when this is the case, we have to deal with what Carswell has termed melanoma. The excessive secretion from the blood of black pigment accompanies the normal pro-

[1] See Reports, 1851–52, p. 253.

cess of involution, tubercular disease and cancer; and, as we have already had occasion to observe, appears to be mainly due to the interference with the oxygenation of the blood. Carswell himself admits the complication of true melanosis with fibrous, carcinomatous, and erectile tissues, and since it does not in itself offer any characteristic features which would serve to establish its pathological identity as an independent formation, we are justified in regarding it rather as an accidental addition than as an essential constituent of a physiological or pathological tissue.

CYSTS.

The formation of cysts in the lungs is of rare occurrence and perfectly latent, so that they are not discovered until after death, unless they excite irritation; they may then find their way into the bronchi and be expectorated. They occupy the lower lobes of the organs; they consist themselves of a double membrane of a clear pellucid appearance, which, under the microscope, present an homogeneous, delicately laminated structure. The laminæ form parallel lines, so as to resemble the pages of an open book. The pulmonary tissue adjoining the cyst is covered by a dense membrane, so that, although entirely surrounded by the pulmonary parenchyma, there is not in reality any real intimate relation with it. They generally contain a limpid fluid, and present an endogenous development of hydatids of the same character as the parent cyst. They vary in size, but an instance which occurred to us of an acephalocyst, sufficiently capacious to contain a hen's egg, must be looked upon as unusually large. It neither contained secondary hydatids nor echinococci. A unique case of cysts in the lungs, filled with air, is quoted by Hasse.[1]

[1] Pathological Anatomy, Syd. Soc. Ed. p. 337.

CHAPTER XXXI.

PLEURITIS.

The serous sac inclosing the lungs and serving to facilitate the movements of respiration, is more prone to morbid affections than any other serous membrane; of these, inflammation is the most frequent, and one that arrests the physician's attention very commonly both in the patient and in the dead subject. Some of the products of inflammation were formerly set down to physiological causes, owing to their being frequently met with in individuals whose histories did not give evidence of pleuritic inflammation having occurred in the course of their life. But the inference is not just, because even in severe pleurisies the symptoms are not necessarily of a character to attract the patient's attention, and most persons are familiar with the occurrence of occasional pains, of a not very enduring character, which may be accompanied by some effusion, though not of sufficient intensity to interfere with the function of respiration. The great frequency of the concurrent inflammation of the pulmonary tissue and its investing membrane, has given rise to a frequent misapplication of the term, and to a variety of theories in reference to the cause of pleurisy. So distinguished an author as Portal attempted to prove that pneumonia was not essentially different from pleuritis; but since the more careful prosecution of morbid anatomy, and the clearer distinction of symptoms during life which we owe to auscultation, no doubt exists that the two, though often associated, differ in their symptomatology as they do in their etiological and pathological relations.

The first stage of inflammation of the pleura is manifested by the appearances of greater or less congestion, causing a multitude of vessels, not visible in the perfectly healthy pleura to the naked eye, to become filled with blood; a marked distinction may sometimes be observed between the venous and arterial channels, as exhibited in the different colors of the two systems. The vessels form an irregular network, and the more intense the inflammatory condition, the more uniform the redness becomes. At times, we find the character of the congestion to be more punctiform, and to resemble, as Laennec has it, an attempt made to dot over the pleural surface with a paint-brush, with small spots of blood of an irregular shape, and closely approaching one another; it is probable that in many cases this appearance is due, not to the peculiarity of the disease, but to a partial emptying of some vessels as a post-mortem effect. The membrane, at the parts most affected, soon loses its natural transparency and gloss, in consequence of a secretion from

the overcharged vessels investing its surface with a coating of lymph or fibrin, a straw-colored semi-gelatinous effusion which may be easily

Fig. 200.

Straw-colored lymph, coating the lower lobe of an inflamed lung, in recent pleurisy, before there was a trace of adhesion to the costal pleura. The outline represents one of two coils of new vessels, seen under the microscope in the fringe of lymph at the lower end.

stripped off from the serous membrane. The microscopic appearances of this fibrin are delicate linear fibrillæ, of a generally parallel direction, enveloping and entangling the granular and corpuscular forms observed in fibrinous exudation. We very rarely meet with cases of what Andral has termed dry pleurisy, as the effusion is a rapid sequel of the first stages of inflammation; but we frequently have an opportunity of observing a limited plastic exudation at one point, while the greater part of the remaining pleura or its fellow may exhibit the first stage of the disease. In the recent cases of sthenic inflammation, the effusion presents the appearance of a thin layer of thick cream, which, at the most dependent parts, seems to be dropping from the organ. The older the effusion, the more it assumes a membranous character, the friction and compression to which it is subject giving to it an irregularly honeycomb or cellular appearance, or causing it early to put on a filamentous or mossy form. The plasticity of the effusion is in a ratio with the plastic character of the blood; hence, it does not always present the characters just described, but varies much in cohesion, in color, and quantity, according to the constitution of the individual attacked, and according to the

Fig. 201.

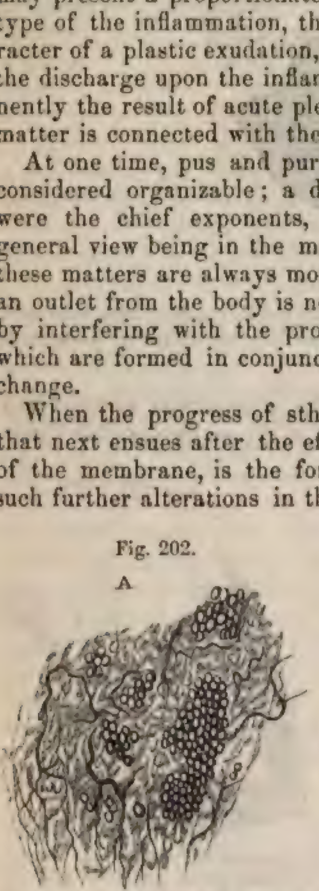
Portion of the lower lobe of the left lung of a patient, compressed by turbid serum, occupying the pleural cavity. A thick layer of lymph covered the hepatized portion of lung; it was perfectly smooth from the contact with the liquid, and there was a free scalloped margin at some parts, of an inch in breadth. The exudation-matter consisted of filamentous matter, entangling corpuscular fibrin.

exciting cause; thus we may, even in the same subject, meet with different products of inflammation at different parts of the same lung. The exudation may be of a more serous or of a purulent character, in which case the sac will contain more or less of these fluids in which we find portions of lymph detached from the pulmonary surface floating about, while their peculiar color, from an admixture of more or less blood, may present a proportionately reddish tinge. The more asthenic the type of the inflammation, the more the effusion departs from the character of a plastic exudation, the more inorganizable it becomes. While the discharge upon the inflamed surface of organizable material is eminently the result of acute pleuritis, the effusion of aplastic or cacoplastic matter is connected with the chronic forms.

At one time, pus and puriform secretions from serous surfaces were considered organizable; a doctrine of which Villermé and Dupuytren were the chief exponents, but which is now entirely exploded, the general view being in the main that advocated by Dr. Hodgkin:[1] that these matters are always more or less excrementitious, and that, where an outlet from the body is not afforded, they invariably retard the cure by interfering with the progress of organization in those substances which are formed in conjunction with them, and are susceptible of this change.

When the progress of sthenic pleuritis is uninterrupted, the change that next ensues after the effusion of coagulable lymph on the surface of the membrane, is the formation in the former of new vessels, and such further alterations in the exudation itself as to induce an assimilation to surrounding textures, and a restoration to a state of comparative, if not absolute, health. The adventitious membranes that thus become permanently formed are of greater or less extent, and may be limited to the one pleural surface, or connect the pulmonary and costal pleura; thus giving rise to further important changes of structure, which may seriously involve the entire thorax, and consecutively even affect the spinal column. The adhesions are of an opaque, whitish hue, and become more firm the older they are. The great frequency with which they are met with, has given rise to their being called ligaments of the lung, as if they formed a normal constituent of the organ. The manner in which the new vessels, that we at a very early stage perceive in the exudation, are produced, has been the subject of much discussion, the arguments adduced being based upon physiological experiments or direct observations, according to the inquirer's bias. The course of the new vessels is generally less tortuous, and presents more parallelism than the vascular channels of the pleura;

Fig. 202.



The lymph of pleuritis, with new vessels already formed in it: a deposit of fat has also taken place. Magn. 20 di.

[1] Lectures on the Morbid Anatomy of the Serous Membranes, vol. i. p. 42.

they appear to be active agents in effecting the absorption of a portion of the exudation matter, and after awhile a retrograde process ensues, and they in part cease to exist. The earliest trace of vessels which we have ourselves discovered in a case of acute pleuritic effusion occupying the base of one lung, where, as yet, no adhesions had taken place, and the creamy effusion was yet eminently fibrinous, exhibited the appearance of small coils, near the edge of the lymph, closely resembling a renal Malpighian tuft, into which two vessels could be seen entering; the sharp outlines of the formations, and a somewhat lighter tinge than the surrounding fibrin, alone marked them; they contained no blood corpuscles, and it was, therefore, only by inference that they were concluded to be young vascular channels. Dr. Hodgkin's view is that the new vessels are formed by the minute bloodvessels of the inflamed part becoming distended, and that their delicate parietes, and the structure through which they ramify become softened, and, yielding to the pressure of the blood in the distended vessels, give way at numerous minute points; he considers that the very small quantity of blood thus escaping from its vessels does not diffuse itself, but is received into the soft substance of the false membrane, which accordingly exhibits numerous bloody points on the surface when detached from the serous membrane. That the *vis à tergo* of the general vascular current is an element in the distribution of the blood to the false membranes, cannot be doubted; but we must demur to the view that it is diffused at random into the exudation matter, and then, as it were, prepares its own channels. The thickness of the false membrane varies from a delicate film to many lines; it may itself become the seat of secondary inflammation, but in most cases it exerts a repulsive influence upon fresh attacks, and assists in protecting the adjacent parts from encroachment. A marked instance of this occurred under our observation lately, in an old man who had suffered a fracture of seven ribs of the left side, followed by inflammation of the corresponding pleura. The two surfaces of the upper half of the pleural sac had formed intimate adhesions, and the subjacent pulmonary parenchyma had remained in a healthy condition; the lower half contained two and a half pints of turbid serum; there was a thick layer of false membrane on the corresponding half of the lung, and this part of the organ was rendered unfit for respiration by the inflammatory process transmitted to it.

The amount of liquid effusion resulting from pleurisy varies from the smallest appreciable quantity to as much as twelve pints. A case, in which this amount was removed from a man aged 33, by paracentesis thoracis, is given by Dr. Novarra, in the *Medical Repository* for 1820. Dr. Hodgkin[1] details an interesting case of empyema, which partly discharged itself through the bronchi, and in which, after death, three large basins were filled with the sero-purulent contents of one pleural sac.

Liquid effusion necessarily induces not only compression of the lung on the affected side, pushing it upwards, but also displaces the adjoining viscera; the heart is forced over to the right thorax when the effusion is into the left pleural cavity; if into the right, the liver is depressed;

[1] Lectures on the Morbid Anatomy, &c. vol. i. p. 121.

in both cases the diaphragm is forced down, and its movements interfered with, while the intercostal spaces of the affected side exhibit a marked prominence, and the ribs are maintained in an elevated position. These anatomical features of extensive pleuritic effusion can scarcely be too strongly insisted upon in the bearing they have upon diagnosis. However, we must bear in mind that exceptional cases occur, in which, owing to a partial reabsorption of the fluid having been effected, the symptoms above detailed are not always so marked as to render the diagnosis easy. Laennec observes that, at the epoch at which we ought to operate, the affected side, though full of pus, is less than the healthy side, owing to this circumstance and the consequent falling in of the parietes.

The secondary changes resulting from firm adhesion being formed are of a different character. If they have been associated with previous extensive effusion, which has become absorbed, the degree to which the compressed lung will regain its former functions depends upon the duration of its confinement, and upon the firmness of the adhesions. The immediate consequence of the absorption was first shown by Laennec to be a falling in of the affected side, owing to the expansion of the lung not taking place in the ratio of the removal of the liquid; and the firmer the adhesions are at given points, the more will this tendency be promoted by their increasing density and contraction. The depression is generally most marked at the lower part of the thorax, about the seventh and eighth ribs; and, owing to the consequent atrophy of the respiratory muscles of the affected side, the equilibrium is destroyed, and the spinal column is deprived of its symmetrical support; from this a curvature of the spine results, the convex margin of which is directed towards the healthy side. The shoulder of the diseased side sinks in proportion. A falling in of the upper portion of the thorax, or of the infra-clavicular region, is commonly noticed as an accompaniment of phthisis, consequent upon the formation of extensive cavities, and the coincident thickening and contraction of the pleura, to which we have alluded when speaking of tubercular disease of the lungs.

There is no affection with which tubercular phthisis is so commonly associated as adhesions between the pleural surfaces, and the relation the two bear to one another appears to be in the ratio of the extent of the former. Hence, the pleurisy has both a chronic and a more local character, and must be set down to the secondary irritation arising after the deposit has been effected. The gradual thickening assumes a cartilaginoid consistency, and the union becomes so intimate that considerable force is often required to remove the lungs, and it is scarcely effected without laceration of their tissue. The intercurrent pleurisy accompanying tubercular disease of the lungs is the source of those flying pains which, from time to time, attack phthisical subjects. The frequency of its occurrence is best illustrated by the statement of Louis, that, in one hundred and twelve phthisical subjects, he found but one whose lungs were perfectly free in every point of their surface. In eight cases only, he found the right pleura wholly unattacked, and in seven the left; in these cases there were either no cavities in the non-adherent lung, or they were very small. Irritation, proceeding from other parts, may equally *give rise* to partial pleurisy; thus, we find it limited to the diaphragmatic

surface, in relation with a diseased liver, spleen, or peritoneum. Irritation of the mediastinal portion may be excited by morbid affections of the heart or bronchial glands; a limited effusion is frequently observed connecting the interlobular fissures, especially in connection with pneumonia and tubercular irritation.

The proclivity to pleuritis is greatest about the middle period of life, and diminishes in the ascending and descending scale of age. Before the fifth year, it is not often met with. Dr. West observes that acute idiopathic pleurisy, unconnected with pneumonia, or in which the inflammation of the lung bears but a very small proportion to that of the pleura, is certainly an uncommon affection during the first years of childhood, and as a cause of death its rarity is extreme; and it certainly has appeared to us that, in cases of pneumonia, there is decidedly a less tendency, in early life, to excite pleuritic inflammation than we should observe in corresponding affections of a later period. With regard to chronic pleurisy, Dr. West remarks, "that, while it is a very rare occurrence as a purely idiopathic affection in early life, it is one of the most common complications of the dropsy which often succeeds to scarlatina."

We do not generally find extensive pleurisy affecting both sides at the same time, while there is a marked difference in regard to the tendency of either pleura to inflammatory attacks, the left side presenting a much greater proclivity than the right. Hasse certainly states that the two sides are equally prone, but considers the fatality to be greater when the left side is attacked, than when the inflammation affects the right. The thirty-five fatal cases which he observed, were distributed in the following manner: nine were double pleurisies, and in five out of the nine, the affection was trifling on one side; in the remaining twenty-six, the left side was the seat sixteen, the right ten times. He also quotes Mohr's experience, who found that of fifty-six cases, the left side was the seat thirty-seven times; the right, nineteen times. Drs. Hamilton Roe, Hughes, and Copland, are also of opinion that the disease exhibits a much greater frequency on the left than on the opposite side of the thorax. It appears from the observations of Messrs. Rilliet and Barthez that the converse is the prevailing character of infantile pleuritis, and that in children the right side is more liable to the idiopathic affection than the left.

EMPYEMA.

We have stated above that the more chronic the form assumed by inflammation of the pleura, the greater the tendency to liquid effusion. Some authors assume that, in these cases, the plastic matter first thrown out may be subsequently converted into pus, a point which has not been determined by direct observation, and certainly is not sufficient to account for the enormous accumulations that sometimes take place. The chronic forms are frequently marked in such a way as to deserve the term of *latent* pleurisy; the symptoms, in the first instance, apparently indicating disease in an organ unconnected with the thoracic cavity. It is here that, during life, the value of the stethoscope is particularly

manifested, as its application will at once remove the difficulties of diagnosis. It is to the chronic effusion of a serous or sero-puriform fluid that the name of empyema is properly applicable, and the inorganizable nature of the pleural contents in these cases is dependent upon diminished tone and vigor of the constitution. The solid matter, according to the views expressed by Dr. C. J. B. Williams, "is thrown out in a disintegrated state, utterly insusceptible of organization, and diffused through the fluid in flakes or particles, forming a mixture more or less resembling pus, although in many instances this is the result of a more chronic form of pleurisy than that which forms lymph, and owes its increase and persistency to the want of vitality in its solid matter. Yet we do meet with cases of empyema which arise from very acute forms of inflammation. In these instances the fluid is more strictly purulent, the solid matter being in the form of globules like those of pus, and seems to be the result of what may be called a suppurating diathesis, in consequence of which all the albuminous products of inflammation tend to assume a purulent character." A marked difference exists between the pleura and peritoneum in regard to this point, for, while the former is peculiarly liable to effusions resulting from a low form of inflammation, the fluid accumulations that we meet with in the latter are more often of a mechanical origin; hence, the result of operative interference is very much more favorable in cases of empyema and inflammatory hydrothorax than in ascites; in the former, when the diseased condition giving rise to the effusion has subsided, there does not exist a tendency to repeated accumulation as in the latter; hence, paracentesis thoracis is more likely to prove a curative agent than tapping of the abdomen, where it rarely serves otherwise than as a means of palliating urgent symptoms. Dr. Hamilton Roe, who has disproved Laennec's statement, that paracentesis was rarely successful, obtained a recovery in eight out of nine cases of empyema, and of nine out of thirteen of inflammatory hydrothorax; and in the same paper from which we derive this information,[1] we find that Mr. B. Philips records a brief analysis of 122 cases of paracentesis, thirty-one of which were performed for empyema, and nine for hydrothorax; of the former twenty-six, of the latter six were cured.

It is, however, admitted on all hands, that it is essential to the success of paracentesis thoracis that it be performed at an early period of the disease.

When the fluid is not evacuated by an operation, it is occasionally discharged spontaneously, either by perforation of the pulmonary tissue, by the thoracic parietes, or by the diaphragm. Dr. Williams considers the second, while Laennec and Hasse look upon the first as the more frequent occurrence. The perforation of the intercostal spaces takes place, not as would be expected, at the base of the lung, but about the middle lobe of the lung; the discharge externally generally being effected by sinuous openings burrowing under the integuments. The prospect of recovery is greater here, than when perforation of the pulmonary pleura leads to an effusion into the lung; for, in the latter

[1] Medico-Chirurgical Transactions, vol. xxvii.

instance, in addition to the mechanical influence of the fluid filling the bronchi, we have to deal with the contamination of the system likely to result from the decomposition of the fluid produced by its contact with the atmosphere.

PNEUMOTHORAX.

This forms one of the modes by which pneumothorax, or the accumulation of air in the pleural cavity, is produced. The most frequent origin of this condition, however, is perforation of the pulmonary pleura by the extension of a tubercular cavity, before the opposing surfaces have become agglutinated by fibrin; the mere softening of one or two tubercles formed close to the pleura, and communicating with a minute bronchus, is described by Dr. Copland as another, though rarer cause of this accident. It has been found to result from rupture of emphysematous vesicles; and Rokitansky also states that it may be consequent upon perforation of the diaphragm or of the mediastinum, arising from acute softening of the stomach or œsophagus. That perforation of the superficial parts leading to the pleural sac induces pneumothorax, will be naturally inferred at once; but it is very improbable that it is ever due to the evolution of gases from the fluids of hydrothorax by spontaneous decomposition, until after death. The perforation leading into the pleural sac is generally a small oval aperture, or a mere fissure, a few lines in length, and situated in the vicinity of the third and fourth ribs near the axilla. The left side offers the greatest liability; Louis found it affected in seven out of eight cases; Hasse has met with nine in which the left, and seven in which the right side was the seat of the lesion; and of fifty collected by Reynaud, thirty-three were on the left and seventeen on the right side. The immediate result of the perforation is imminent, if not actual suffocation from collapse of the lung; if death does not at once ensue, intense pleuritis is set up, and the patient rarely survives for many days. We find the lung compressed to the utmost, and the other viscera are much displaced, according to the side in which the air has accumulated, the epigastrium protruding from the descent of the diaphragm, and its action on the liver and stomach; the other pathological conditions are those indicating inflammation of the pleura; and we may observe an attempt at repair in the shape of a false membrane investing the fissure. The occurrence of pneumothorax is only possible when the pleura at the point of perforation has not previously become adherent; but it sometimes happens that the ulcerative process is continued after the pulmonary and parietal pleura have been agglutinated, and passing through the uniting medium attacks the intercostal muscles and integuments. A fistulous opening may thus be established.

HYDROTHORAX.

Hydrothorax, or dropsical accumulation of fluid in the pleural sac, occurs in two forms, as a primary and as a consecutive lesion; the former is a disease of much less frequent occurrence than was at one time

supposed. Laennec states that one could not establish a higher ratio for the occurrence of idiopathic hydrothorax as a cause of death, than one in two thousand. Let us hope that the days are past in which such errors of diagnosis, as he alludes to, can be committed; for he asserts having found hypertrophy of the heart, aortic aneurism, phthisis pulmonalis of a somewhat irregular character, and even scirrhus of the stomach or liver, without the least effusion into the pleura, mistaken for hydrothorax. The affection consists in the effusion of a limpid serosity into the pleura, to a greater or less amount, generally limited to one side, and unaccompanied by any appreciable change of structure in the serous membrane; the compression exerted by the fluid upon the lung and the adjacent parts, is necessarily the same in this instance as in those forms of fluid accumulation which have already been considered.

The secondary or symptomatic form of hydrothorax is a common sequel of acute or chronic diseases, heralding the fatal termination, and giving evidence of that loss of tonicity that results from exhaustive maladies. The circulating system is more frequently found to be at fault than any other; hypertrophy of the heart, valvular disease, pericarditis, are common causes; it is also often associated with renal degenerations, tubercular and cancerous affections. The same cause that induces the effusion into the pleura, gives rise to dropsical accumulation in other serous cavities, or in the cellular tissue; hence symptomatic hydrothorax is generally accompanied by other affections of the same kind. As in the primary form, we find no definite lesion of the serous surface associated with it; it is not in fact an affection of the membrane at all, but exclusively of the vascular system, and we must look to the blood and the capillaries for an explanation. According to Laennec, it rarely occurs more than a few days before death; and though it often produces no sensible effect upon the patient's feelings, it often causes suffocative attacks, which render his last moments painful amd distressing. The liquid itself is commonly a clear straw-colored serum.

HÆMOTHORAX.

When the pleural sac is filled with blood, or with a fluid of a decidedly sanguineous character, we have to deal with hæmothorax. This is commonly due to some mechanical lesion or to the rupture of an aneurism; but it has also been met with as the result of capillary exhalation—of the same character as that to which active or passive spontaneous hemorrhages are commonly attributable. It is said to be capable of reabsorption or to superinduce inflammatory action; or, again, to be liable to decomposition, and thus to give rise to pneumothorax.

GANGRENE.

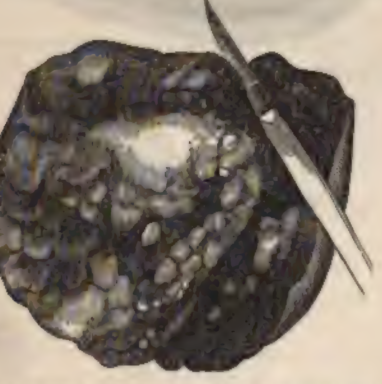
Before considering the adventitious products met with in the pleura, we must briefly advert to the occurrence of gangrene. It is an unusual lesion, and is commonly connected with gangrene of the pulmonary

tissue. The sloughs are to be recognized by their greenish brown or blackish hue, of a circular or irregular form, extending to some distance beyond the part detached. The fetid smell will also assist in determining the character of the lesion.

ADVENTITIOUS PRODUCTS.

Among the homologous formations occurring in the serous membrane of the lungs, authors enumerate cartilage, bone, and fat. A cartilaginoid thickening of the pleura, more especially at and about the apices, is by no means unusual; but the microscope invariably resolves this deposit into one of a fibrous, or, as Lebert terms it, chondroid, character. If the result of gone-by inflammation of the pleura, it affects the free surfaces of the membrane with which it intimately coalesces; when due to subserous congestion, it is found in the subserous tissues, and in the membrane itself. The lung exhibits fibroid formations, which, like those found on the heart, the liver, or the spleen, are frequently but the indications of a fibrinous blood crasis, independent of any actual inflammatory process; they are smoothed, nodulated, very dense, and adherent only by their exterior surface. We meet with the formation of true bone in the pleura as little as of genuine cartilage: the osteoid deposits are mere amorphous aggregations of calcareous matter, occurring in plates or irregular points; they may be encysted, and occasionally they form pedunculated projections invested by the pleura. To this class we

Fig. 208.



Old cartilaginoid capsule of the apex of a lung, in a man aged 63; both lungs were similarly affected, and like patches were also found on the spleen. There was some appearance of obsolete tubercle, and much black pigmentary matter. No definite structure was to be traced in the capsule by the microscope.

may probably refer the case of ossification of the lungs given by Dr. Baillie,[1] for, in the delineation, the ossified parts are distinct polypoid offsets from the pulmonary tissue, though apparently invested by the

[1] Morbid Anatomy, Second fascic. pl. vi.

same membrane. Rokitansky states that fibrous exudations invest the costal as well as the pulmonary, but that they only ossify on the costal pleura, the subserous products occurring chiefly in the intercostal spaces, from which they may be discharged into the cavity of the thorax in the shape of round nodules. That ossification of the pleura is generally preceded by some inflammatory condition, may be inferred both from the frequent occurrence of other inflammatory products in the pleura, and from the analysis of a considerable number of cases in a dissertation by Dr. Posselt;[1] he found that out of twenty-seven instances twelve affected the right, and fifteen the left side, while the ratio of the sexes was as thirty men to four women. The size of the osteoid deposit is occasionally very extensive. Dr. Hodgkin removed from an old man who died at Guy's Hospital, a plate of bone subjacent to the parietal pleura, which half encircled the chest and formed a considerable mass. The fatty deposits which we find connected with the pleura are rarely on the free surface, but seem to be the result either of a transformation of previous inflammatory products, or a fatty growth under a false membrane. Recent fibrinous exudation occasionally closely resembles

Fig. 204.

Fig. 205.

Naked-eye and microscopic view of cancer of the pleura. The growths were mainly in the interlobar fissures, and occurred in a female who had malignant disease of the left mamma. The liver also exhibited cancer-nodes.

adipose tissue both in color and form, though there can be no real difficulty in determining its nature. Tubercular deposit is not often met with in the pleura; and almost exclusively occurs as a secondary form

[1] De Pleuræ Ossificatione, Heidelberg, 1839.

of the disease. The pleura, in this respect, differs in a marked manner from the peritoneum and the arachnoid, both of which are more prone to primary tuberculosis than the former. Tubercle forms either under the pleura or in the sac—in the latter case, its seat is invariably in a false membrane; tubercles of this description, Dr. Hodgkin remarks, when thickly set, have been mistaken for thickening of the pleura itself. Pleural tubercle, according to Rokitansky, not unfrequently softens and gives rise to tuberculous abscesses in the different pseudo-membranous structures in which it is deposited; these abscesses may penetrate the pleura, and even the thoracic walls.

Malignant growths never affect the pleura primarily, but involve the membrane by extension from the mamma, the bronchial glands, the mediastina, or other neighboring tissues. They appear on the pleura as flattened masses, rarely larger than an almond, surrounded by a halo of bloodvessels. While tubercle occurs in very numerous small spots, spread all over the membrane, cancer is only seen in a few isolated patches. Both are liable to induce serous effusion of a sanguinolent character. Medullary carcinoma and melanotic cancer are the forms of malignant disease that most frequently attack the pleura.

Hydatid cysts appear to occur in the pleura. Cruveilhier[1] details a case in which a large number of acephalocysts were discharged from an artificial opening, apparently communicating with the pleural cavity, in a whitesmith, aged 29; the man entirely recovered, after the evacuation of above five hundred hydatids. Dr. Hodgkin,[2] in his fifth lecture, speaks of a specimen presented to Guy's Museum, in which a large cyst containing acephalocyst hydatids is situated in part beneath the close pericardium about the base of the heart, and partly under the pleura pulmonalis, at the root and summit of the right lung.

[1] Anatomie Pathologique, vol. i. p. 247.
[2] Morbid Anatomy of the Serous Membranes, vol. i. p. 187.

THE PATHOLOGICAL ANATOMY OF THE ALIMENTARY CANAL.

CHAPTER XXXII.

I. OF THE MOUTH AND FAUCES.

CONGENITAL malformations sometimes consist in an excess of the natural number of parts, so that the jawbones, "the mouth, and the tongue, are double, and unite in one common gullet." More often they show themselves by defective formation of the mouth and fauces (*astomia*), of the upper jaw (*ateloprosopia*), of the lower law (*agnathia* and *atelognathia*), of the lips (*achelia* and *atelochelia*), of the tongue (*ateloglossia*). Arrest of development shows itself in the common single or double harelip, the fissure existing at the union of the intermaxillary with the upper jawbones; in fissures of the hard and soft palate; in fissures of the tongue, the lower lip, and the lower jaw, which are all very rare. In some rare instances, the orifice of the mouth is wanting (*atresia oris*).

The buccal mucous membrane shows but little tendency to be affected by catarrhal inflammation; it is, however, often inflamed in one or more spots from some local irritation, or morbid action. Thus, a carious tooth, a piece of diseased bone, a crop of ulcerations, will excite inflammation in their vicinity. In adults, general inflammation of the mucous membrane is sometimes produced by the abuse or excessive action of mercury, and shows a marked tendency to pass into a state of ulceration and sloughing. Mr. Tomes mentions the occasional occurrence of spontaneous salivation, with considerable inflammation of the gums. Chronic inflammation of the gums is not uncommon, and has appeared to us sometimes to be of rheumatic origin. It may extend over the whole mouth, or be confined to the vicinity of two or three teeth. "The surface of the gums," Mr. Tomes says, "becomes minutely nodulated; and the secretion of epithelium increased; the papillæ are increased in prominence, while the substance of the gum is generally thickened, and the edges about the teeth become thick and round."

In another form of so-called chronic inflammation, the gum rather decreases in size, and "assumes a very smooth and polished surface, and mottled aspect;" the hard palate also becomes implicated, and there is acute intermittent pain. Ulcerations often form on the gums, as well

as on other parts of the mucous membrane of the buccal cavity; they are sometimes simple aphthæ, sometimes small, round, slightly excavated, and without any surrounding inflammation. In some cases, the ulcerations are attended with much inflammation, and swelling of the mucous membrane and subjacent tissues, and, in others, they are rather of a sloughy nature, and form upon a surface dark colored by asthenic congestion.

Epulis (ἐπί, οὖλον) is a fibrous tumor, which originates in the fibrous tissue of the gums, or in the periosteum, and not only grows outward toward the cavity of the mouth, but also penetrates into the Haversian canals and cancelli of the bone. Its surface is generally pretty smooth, "like the gum," or it may be rough and more or less lobulated. "Osseous spiculæ not uncommonly shoot," according to Mr. Tomes, "from the surface of the jaw into the tumor, and, in some cases, isolated nodules of bone (calcification?) are found in the substance." *Polypus* of the gum is a local hypertrophy of its tissue, occasioned by some mechanical irritation. It shows, "on section, an undulating fibro-cellular tissue, covered by a thick layer of epithelium." *Vascular tumors*, consisting essentially of dilated vessels, sometimes appear on the gums. Mr. Tomes describes one of a bright scarlet color, soft in texture, and easily compressed and emptied of blood, and prone to bleed on slight irritation. *Cancer*, almost always in the form of scirrhus, occasionally attacks the gums. Its size varies usually from that of a pea to that of a nut. It ulcerates after a time, and may throw out fungous growths.

The vesicles of herpes, and the pustules of variola, occasionally are developed upon the buccal mucous membrane.

The *croupy process* (the diphtheritis of Bretonneau) appears in adults, according to Rokitansky, in two forms. In one, "after a previous vivid or dark purple reddening of one or more papillæ, and the vesicular elevation of the epithelium at the point and the sides of the tongue, dots or patches, of the size of a lentil or pea, appear on the inner surface of the lips and cheeks, and, finally, on the mucous membrane of the fauces. They present an exudation which has a frosted, or flocculent, or villous appearance, or is more of a membranous character, and extends into the cavities of the follicles; it is of a grayish, or yellowish-white color, and of a lardaceous, or soft, creamy, or fluid consistency; if removed, a shallow, excoriated depression, surrounded by an inflamed margin, remains, on which the exudation is repeated, involving a further destruction of the mucous tissue. In the second instance, livid spots, which rapidly coalesce, and become invested with a dirty, gray, shaggy, pultaceous, and sanious exudation, form upon the softened, bleeding gums, and the mucous membrane of the cheeks, the fauces, and the tonsils. The gums themselves ultimately degenerate into a bad-looking, pulpy, sanious mass, and the mucous membrane of the cheeks and fauces, underneath the exudations, is equally found converted into a friable fetid pulp, or a firm slough." The epidemic adynamic character of the above described process, is now well known. Andral, writing more than twenty years ago, contended most justly that the congestion of the part affected, though first in order, was but secondary in regard to casual agency; and Rokitansky's investigations of the different kinds of intra and extra-vascular

fibrin, which we have before noticed, have made it almost certain that the essence of the disease consists in an alteration of the liquor sanguinis, which gives rise to unhealthy exudation from the bloodvessels at various parts. Andral mentions that blistered and all denuded surfaces, during the prevalence of such epidemics, become covered with false membranes like those which form on mucous surfaces. Wounds and ulcers at such periods are found unusually inapt to heal, and all experience of the *juvantia* and *lædentia*, seems to testify that the local morbid process is extremely different from common inflammation, and is essentially dependent upon a grave alteration of the general system. The color of the exudation, which is naturally whitish, is often rendered darker by sanguineous effusion saturating it; when this is the case, its aspect, and the extreme fetor which it exhales, give to it a considerable resemblance to a gangrenous slough. This would be still more increased if it were situated, as it sometimes is, beneath, and not upon the layer of epithelium.

The succeeding kinds of ulceration which we shall describe are, for the most part, seen in children. *Aphthæ* are small whity specks, sometimes so closely set together that they coalesce and form patches, which may be very extensive. They separate after a time, leaving the mucous membrane beneath either simply excoriated, or superficially ulcerated. After being detached they are often produced again, and this may occur several times in succession. It is not yet quite decided of what these specks really consist; analogy would support the olden opinion of their being simply a variety of false membrane, but some microscopists contend that they are solely clusters of parasitic fungi. We incline with Dr. West to the belief that the former opinion is the more correct, not that we doubt the correctness of the observation of a growth of fungi in the exudation, but that we think it much more probable that these are developed secondarily in an unhealthy, aplastic, animal matter, which is freely exposed to the contact of air. Dr. West suggests the idea that the sporules of this fungous growth might, by lighting upon the mucous membrane, and exciting irritation there, cause the production of the aphthous specks. This, we think, is very improbable, as, were this the case, but few children could hope to escape: the diffused sporules which affect one child, might as well affect all who were at all disposed. It should be mentioned that the term *muguet* is applied by the French to the more extensive deposits of this kind. Bad health, indigestion, or abdominal disorder are the precursors and attendants on aphthæ. The whole of the buccal mucous membrane appears to be in a state of asthenic inflammation, and the same condition extends in some measure to the whole alimentary track. The disorder is, certainly, a general one, manifesting itself by a local symptom, and not confined to that part. Adults are sometimes affected by aphthæ as the result of indigestions, or as indicative of decaying vital powers. A late eminent physician prognosticated his own approaching decease from the appearance of aphthæ on his tongue. Dr. H. Salter describes small circular ulcers which form at the tip and along the edges of the tongue. These we have experienced ourselves, and can scarce think they should be separated from aphthæ. According to him they are produced by the

effusion of lymph into one of the fungiform papillæ, which soon disappears by sloughing or ulceration, leaving an ulcer which continues to spread for some time.

Follicular stomatitis is described by Dr. West as sometimes idiopathic, sometimes a concomitant of measles. In either case it is rare, after five years of age. "The mouth is hot; its mucous membrane generally of a livid red, while a coat of thin mucus covers the centre of the tongue. On the surface of the tongue, especially near its tip on the inside of the lips, the cheeks, near the angles of the mouth, and less often in other situations, also, may be seen several small, isolated, transparent vesicles on the ulcers, which, after bursting, they leave behind. The ulcers are small, of a rounded or oval form, not very deep, but having sharply cut edges; and their surface is covered by a yellowish white, firmly-adherent slough." "When the ulcers are healing, no change in their aspect is observable, and they continue to the last covered by the same yellow slough, but, by degrees, they diminish in size; and seldom or never is any cicatrix observable in the situation which they occupied." The vesicles form in crops, not generally containing many; the resulting ulcers sometimes coalesce and form a continuous patch. The affection is sometimes complicated with herpes of the skin of the lips, and might almost be considered as a similar eruption of the mucous surface.

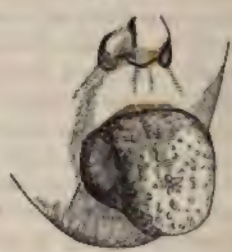
In *ulcerative stomatitis*, as described by the same observer, "the gums are red, swollen, and spongy, and their edge is covered with a dirty white or grayish pultaceous deposit; on removing which their surface is exposed, raw, and bleeding. At first only the front of the gum is thus affected; but as the disease advances, it creeps round the teeth to their posterior surface, and then destroying the gum, both in front and behind them, leaves them denuded, and very loose in their sockets. On those parts of the lips and cheeks, however, which are opposite to, and consequently in contact with, the ulcerated gums, irregular ulcerations form, which are covered with a pultaceous pseudo-membranous deposit, similar to that which exists on the gums themselves. Sometimes, too, deposits of false membrane take place on other parts of the inside of the mouth, the surface beneath being red, spongy, and bleeding, though not distinctly ulcerated. . . . When recovery has commenced, the disease ceases to spread; the drivelling of fetid saliva diminishes; the white, pultaceous deposit on the gums, or on the ulcerations of the cheek or lips, becomes less abundant; the ulcers themselves grow less; and, finally, the gums become firm," and slowly, and perhaps with partial relapses, regain their healthy condition. The disease is common, rarely fatal, rarely associated with, or proceeding to gangrene. It is sometimes designated by the term Noma.

True *gangrene of the mouth* is a much less frequent, and much more fatal affection. It is very seldom idiopathic, almost always occurring consecutively to measles or some other disease. Messrs. Rilliet and Barthez found, out of twenty-nine cases, nineteen aged from two to five years, and ten aged from six to twelve. We again quote Dr. West's description, which pictures very well the only case which we have witnessed ourselves. There is at first scarce any suffering, and some unusual fetor

of the breath, some profuse secretion of offensive saliva, and swelling of the cheek, are the first circumstances which are observed. The characters of the swelling of the cheek are almost pathognomonic. It is not a mere puffiness, but is tense, red, and shining—looking "as if its surface had been besmeared with oil, and in the centre of the swollen part there is generally a spot of a brighter red than that around. The cheek feels hard, and is often so unyielding, that the mouth cannot be opened wide enough to get a good view of its interior. The disease is almost always limited to one side, and generally to one cheek." Occasionally, it begins in the lower lip, never in the upper, but it may extend to either. "Whatever be the situation of the external swelling, there will generally be found within the mouth, at a point corresponding to the bright red central spot, a deep excavated ulcer, with irregular jagged edges, and a surface covered by a dark, brown, shreddy slough. The gums opposite to the ulcer are of a dark color, covered with the putrilage from its surface, and in part destroyed, leaving the teeth loose, and the alveolæ denuded. Sometimes, especially if the disease be further advanced, no single spot of ulceration is recognizable, but the whole inside of the cheek is occupied by a dirty putrilage, in the midst of which large shreds of dead mucous membrane hang down. As the disease extends within the cheek, a similar process of destruction goes on upon the gum, and the loosened teeth drop out one by one. The saliva continues to be secreted profusely, but shows by the changes which take place in its character the progress of the disease. At first, though remarkable for its fetor, it is otherwise unaltered, but afterwards loses its transparency, and receives from the putrefying tissues over which it passes, a dirty, greenish, or brownish color, and at the same time acquires a still more repulsive odor. While the gangrene is thus going on inside the mouth, changes no less remarkable are taking place on the exterior of the face. The redness and swelling of the cheek extend, and the deep red central spot grows larger. A black point appears in its midst; at first, it is but a speck, but it increases rapidly, still retaining a circular form—it attains the bigness of a sixpence, a shilling, a half-crown, or even a larger size. A ring of intense redness now encircles it, the gangrene ceases to extend, and the slough begins to separate. Death often takes place before the detachment of the eschar is complete; and it is fortunate when it does so, for sloughing usually commences in the parts left behind. The interior of the mouth is now exposed, its mucous membrane and the substance of the cheek hang down in shreds from amidst a blackening mass," which exhales a horrible fetor. There is no acute pain throughout, the patient is generally rather drowsy, and death takes place quietly in most cases. No cause has been assigned for the occurrence of gangrene in this part; all that can be said is that a true mortification or death of the textures seem to take place, which is itself the primary evil, and not the result of inflammation, disease of the vessels, or obstruction of their channels. This is a good illustration of the doctrine we maintained, when speaking of mortification, viz: that it essentially consisted in a loss of the vital powers which maintain, in opposition to those of inorganic chemistry, the complex constitution of the animal tissues.

The *tongue* is liable to be affected by inflammation, or *glossitis*, as it is termed. This in some rare cases, said by Dr. Salter to occur most often in scrofulous persons, causes the formation of abscess. On the matter being evacuated, the tongue speedily returns to a healthy state. Sometimes a partial inflammation of the tongue is met with, the morbid process being confined to the portion of the base bounded in front by the V-shaped line of circumvallate papillæ. It occurs as an extension of tonsillitis, which we shall presently notice. Deglutition in these cases is seriously interfered with. The inflammation of the gums, which is produced by mercury, sometimes involves the tongue, and occasions, in some cases, very great and rapid swelling. It does not seem to have even produced suppuration. One variety of glossitis has been distinguished by the term *erectile*, by Dr. Salter. He describes "the morbid condition of the tongue in this disease as consisting in an enormous and rapid distension of the organ by blood, rendering it very large,

Fig. 206.



Tongue, swollen by Glossitis.

hard, and stiff. The distension becomes so great that respiration through the mouth is quite prevented, and even can with difficulty be performed through the nostrils. Though the congestion becomes so intense that the organ is of a dark black color, neither mortification nor abscess appears to have ever taken place. Free incisions give exit to the blood, and recovery ensues. Sometimes one-half of the tongue only is affected. In most cases it occurs in persons who are in perfect health, and without any manifest exciting cause."

Severe and deep *ulcerations* of the tongue may arise "from mere disorder of the alimentary canal," especially in debilitated persons. Some of these, attended with much induration, may bear a very close resemblance to cancerous ulcers. Constitutional syphilis produces small superficial circular ulcers, which sometimes extend in depth, and sometimes in length only. *Rhagades* or fissures result from the same cause: they often occupy the medium line in the front part of the organ; they may be mere cracks, or extend three-quarters of an inch in depth, with irregular ulcerated edges. Often, they are associated with tubercles of the surface of the tongue. These, which are admirably described by Dr. H. Salter, under the name of *glossy tubercle*, appear to be of the same nature as the syphilitic tubercles termed *gummata*. Ricord speaks of them as deep-seated tubercles of the subcutaneous areolar tissue, a

kind of chronic furuncles; and refers to two cases of recurring syphilis, in which the tongue was so full of them that it felt as if stuffed with nuts. According to the French observer, they produce horrible destructive ulcerations. Dr. H. Salter describes them "to consist in an effusion of lymph into the cellular tissue underlying the mucous membrane; this effusion is very dense, and raises and distends the surface of the tongue at the affected part above the surrounding portions; the effect is that the papillæ near it are opened out, and sometimes totally obliterated. Hence the surface of the tubercles is smooth, and, as they become absorbed, the papillæ reappear again.

The tongue is liable to be the seat of cancerous growths of the scirrhous and epithelial species. The former is described by Mr. Travers as at first being an irregular rugged knob, generally situated in the anterior third, and midway between the raphé and one edge. Ulceration sometimes takes place very rapidly; the surface at the same time throwing out luxuriant fungous growths: in other cases it "is very uneven, clear and bright granulations appearing in parts, and in others deep and sloughy hollows." In a peculiarly interesting case of epithelial cancer, carefully watched by Dr. H. Bennett, the first appearance of the disease was a small ulcer on the margin of the tongue. This extended, in spite of its being shielded from the pressure of the teeth, and had hard, everted edges, undermined some way by ulceration. These became more ragged, and here and there over the surface some degree of suppuration and sloughing occurred. Much improvement followed the excision of the tumor—the wound healed favorably. Not long after, however, the glands under the jaws enlarged, and were removed; and, in about nine months after this, the disease returned in the tongue and proved fatal. The morbid growth which had been removed presented, on a transverse section, a tract of white, indurated, convoluted structure immediately below the ulcer, and above the muscular substance of the tongue. This indurated tract was half an inch thick posteriorly, and consisted of a fibroid structure inclosing debris of muscular fibre, and some of the characteristic circular loculi of epithelial cancer. The surface of the ulcer was covered with papillary elevations, which consisted chiefly of enlarged, softened epithelial scales splitting into fibre, so as to form a kind of fringe. This history shows, we think, beyond any doubt, that what Dr. Bennett would distinguish as cancroid, are in many, if not most cases, as true cancers as any of the other species. The circumstance mentioned by Dr. Bennett in his Appendix to his work is curious and significant, viz: that the enlarged glands beneath the jaw contained quantities of epithelial scales similar to those found in the primary growth. This indicates a potentiality in the blastema, absorbed from the epithelial tumor, to cause a reproduction of like cell-structure.

Fatty tumors and *simple cysts* are occasionally met with in the tongue, and Dr. Salter mentions the occurrence of pediculated *polypoid growths*, which seem to be of the nature of fibrous tumors, or, perhaps, in some cases of enchondroma. The tongue is liable to be affected by an extraordinary *hypertrophic enlargement*, in consequence of which it protrudes from the mouth, sometimes as much as two and a half inches. The

structure is altered, becoming much more dense than natural; but it has not been determined exactly in what the alteration consists. In one case, recorded by Mr. Liston, the enlargement of the organ seems to have been occasioned by the development of nævus-like structure. *Atrophy* of the tongue only occurs as the consequence of paralysis, from division of the hypoglossal nerve, or attacks of hemiplegia. It is, of course, confined to the affected side. Dr. Salter gives an interesting account of the morbid changes which the lingual papillæ undergo. The circumvallate papillæ may be hypertrophied, and form little tumors as large as peas. The epithelial caps of the conical or filiform papillæ may become extraordinarily elongated, so as to be half an inch long; they are of a dark color, and look exactly like little brown hairs. Minor degrees of this condition are, we think, not uncommon. The papillæ sometimes become atrophied. "Mr. Lawrence mentions the case of a person, in whom, from habitual drinking, the tongue was, for the greater part of its surface, destitute of papillæ: it was white, smooth, and opaque on the surface." Blood and lymph may be effused into the substance of the fungiform papillæ. The pus which so commonly collects on the surface of the tongue in disease, consists of detached, and more or less disintegrated epithelium, with varying proportions of amorphous matter. We can corroborate Dr. Salter's statement, that, in some healthy persons, the tongue is habitually furred. In very rare cases the frænum of the tongue is so short that it is quite tied down to the floor of the buccal cavity, and cannot perform its proper movements. Minor degrees of the same condition are not infrequent, and gradually improve of themselves. In the opposite condition, "the movements of the tongue are too free; it can be inverted, and its apex thrown back into the pharynx, which embraces it," and thus the access of air to the lungs through the glottis is prevented. The sides of the tongue have been known to become closely adherent to the internal surface of the cheeks.

The tonsils are a more common seat of inflammation and its consequences. In an acute attack they become more or less, sometimes enormously swollen, so as to impede the respiration. The pillars of the fauces, and the soft palate, are also involved in the inflammation. Suppuration often occurs, and is, perhaps, the best result, next to complete resolution; but more frequently, the imperfectly subdued hyperæmia produces actual enlargement, and fresh attacks recurring, a chronic hypertrophy of the gland is the result. We have examined some enlarged tonsils which had been excised, and found their structure to be quite identical with that of the healthy gland, so that the alteration constituted a true hypertrophy. It seems worth while to notice briefly the structure of the tonsils, which we think is not well understood, as it explains in some measure their great liability to hypertrophic enlargement. They are made up of a number of duplicatures and involutions of the mucous membrane, which, however, is differently constituted here to what it is in other parts in the vicinity. A vertical section shows the thin surface layer of scaly epithelium with a thick underlying stratum, consisting of nuclear, or very slightly developed celloid parti-

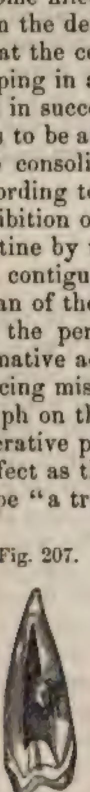
cles. This layer is traversed by vessels, which are of capacious size in hypertrophied specimens, running up to the basement-membrane which supports the layer of scaly epithelium. When there is any habitual hyperæmia, and consequent exudation, this low submucous celloid growth readily assimilates the effused plasma into similar substance, and so the enlargement continually goes on. The morbid condition which most resembles it is enlargement of the Peyerian patches, which we shall presently describe. Induration not unfrequently occurs as the result of inflammation, and depends, beyond doubt, on a fibroid development of the exudation. Rokitansky says: "In scrofulous subjects the tonsils are often affected, in addition 'to hypertrophy and habitual hyperæmia, with a peculiar blennorrhœa, and the purulent secretion not unfrequently becomes inspissated, so as to form tubercular cheesy plugs, or even chalky concretions. These, in their turn, keep up a perpetual state of irritation in the tonsils." Cancerous disease is very rare in this situation, but common indurated enlargement has often been spoken of as scirrhous.

II. MORBID CONDITIONS OF THE TEETH.

The brief summary that we shall give of these conditions is taken from the excellent work of Mr. Tomes on the subject, to which we must refer for fuller information. *Malposition* of the other teeth is scarce more than a disfigurement, but when the wisdom teeth take a wrong direction the effects produced are sometimes very mischievous. Those of the lower jaw cause more serious evil by their wanderings than those of the upper. Sometimes the tooth, though not deviating from its proper position, is held down by indurated gum. Esquirol mentions a case in which mental derangement depended on this cause. The wisdom tooth may take a false direction inward or outward, and cause by its pressure, ulceration of the tongue or the cheeks. It may grow directly forwards against the posterior surface of the second molar, which has proved the source of severe pain, resisting all treatment but that of extracting the offending tooth. Lastly, the tooth may advance against the coronoid process, causing disease and necrosis of the bone, and inflammation and abscess in the surrounding parts. The teeth are very liable to *caries*, which is an affection very much of the same kind as that occurring in bones. Mr. Tomes believes that "the dentine, from abnormal (nutritive) action, loses its vitality," and therewith becomes liable to be decomposed by the fluids of the mouth. It seems necessary that both conditions should exist, that the tissue should be dead, and that the oral fluids should be in an acid state, capable of dissolving it. Test paper applied to carious teeth almost invariably shows the presence of free acid. Healthy saliva is alkaline, while that of dyspeptic persons is prone to be acid, and it is in such that caries is most apt to occur. The enamel is, of course, first affected, but a very small perforation through this tissue may exist with a considerable amount of disease in the subjacent dentine. It appears that when the acid solvent has once

penetrated to the surface of the dentine, it extends laterally under the enamel, destroying, extensively perhaps, the body of the tooth, and undermining and eroding the enamel on its attached surface. The destructive process does not go on nearly so fast in the fang, which seems to possess a higher degree of vitality than the crown. A most interesting observation of Mr. Tomes demonstrates completely the vital nature of the actions going on in the dentine under the influence of disease. He shows that when a portion of dentine has become dead, it is circumscribed by the consolidation of the adjacent living tissue. "The tubes become filled up, they are rendered solid, and the circulation is cut off from the dead mass by the obliteration of the tubes." It is remarkable "that the consolidation does not go on gradually from without inwards, keeping in advance of the decay, but occurs at intervals." It is formed also in successive lines, a second one being produced when the first begins to be attacked, and afterwards a third, when the second gives way. The consolidated zones vary in width and in completeness, probably according to the vigor of the conservative action. Another interesting exhibition of vital action is displayed in the production of secondary dentine by the surface of the pulp, under the excitement of caries in the contiguous tissue. This vascular papilla, originally the formative organ of the dentine, which had for years confined its action to nourishing the perfected structure, under the stimulus of disease renews its formative action, and throws out a barrier between itself and the advancing mischief. How very analogous is this to the throwing out of lymph on the outer surface of a hollow viscus which is threatened with ulcerative perforation! The structure of secondary dentine is not so perfect as the original, and it is commonly vascular. Under the microscope "a transverse section of carious dentine, rendered soft, like cartilage, from the loss of its lime, presents a cribriform appearance. The tubuli are much enlarged and irregular in outline," differing entirely from their normal shape. This indicates that the solvent enters the tubes, and dissolves, first, their walls, and afterwards the intertubular material. In the consolidated zones the deposit obliterating the tubes is first removed, and afterwards their walls and the intervening tissue. A confervoid growth is very often seen on carious teeth, and on the tartar that may incrust them. Imperfect formation of the enamel is a frequent cause of caries. It is mostly deficient on the surface, presenting deep pits, with the intervening structure well developed; sometimes, however, small cavities exist in its substance, while the rest is perfect. Deep narrow fissures are often met with extending from the free surface to within the $\frac{1}{100}$th of an inch of the dentine; the enamel forming the walls of these is in parts perfect, in parts imperfect. Not only may the enamel be deficient, but it may be also imperfectly formed in various parts. The columns of its pulp consist of cells and granules, which, normally, become lost and fused in the homogeneous fibre; sometimes this fusion does not take place, and the granules re-

Fig. 207.

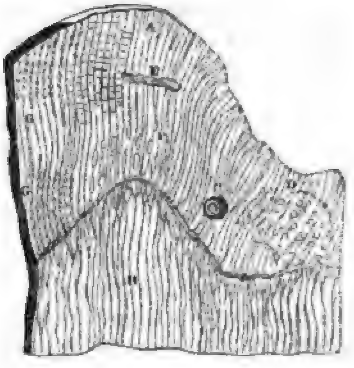


Drawing of a tooth attacked by caries, with barrier of secondary dentine.

main, giving the enamel fibre a permanently granular aspect; or the cells do not undergo their wonted arrangement and elongation, and thus, though they calcify, do not form fibres. Sometimes the fibres of the enamel are not perfectly united at their margins; the resulting interspace may either appear as a broad line, or as a series of minute cells.

The teeth are subject not only to decay, but to death, to *necrosis*, which may be either complete or partial. After this has occurred, certain physical changes commence. "The tooth gradually assumes a darker hue than natural, which increases in intensity till it is almost black. The dental periosteum gradually detaches itself from the fang, the tooth becomes loose, and unless held in by the crooked form of the roots, drops out. The surface of the fangs is generally rough, and,

Fig. 208.



Imperfect formation of enamel. From Mr. Tomes's work.
A. Enamel.
B. Dentine.
C. A perforation in the enamel.
D. A cribriform layer of tissue in the enamel.
E. A large cell lying transverse to the enamel-fibres.
F. Cells in the enamel about the apices of the coronal dentine.
G. Lines of minute cells between the enamel-fibres.

near the neck, dotted over with nodules of hard green-colored tartar, while the ends of the roots often look worm-eaten, as though absorption had commenced." Mr. Tomes compares the process of necrosis of a tooth with that which occasions the shedding of the antlers of a deer. In both, the minute tubes or cavities through which nutrient fluid is conveyed, become obliterated by calcareous deposition; the whole tissue being consolidated into an inorganic mass. The dead tooth acts as a foreign body, causing inflammation and suppuration of the dental periosteum, as well as absorption of the latter, the alveolus, and gums. More serious effects are occasionally produced, "the periosteum of the alveoli becomes inflamed, together with the neighboring parts;" and, if the case be still neglected, the adjoining teeth are not unfrequently lost, and necrosis of a considerable part of the jaw may also result. In some instances, where the death of the tooth has taken place gradually, patches of newly-formed cementum, thrown out by the irritated dental

periosteum, adhere closely to the latter, and thus the tooth is held in its place. A single spot of necrosis in the fangs may cause inflammation, and abscess, and such severe pain that the tooth, though otherwise quite healthy, is obliged to be removed. "Instances are not uncommon when the pulp of the tooth has died while the external surface of the fang has preserved its vitality. In these cases the dentine becomes discolored, and gives a general dark appearance to the tooth. One of the three fangs of a molar tooth may alone be affected by necrosis, or the disease may be confined to one side of a single fang, producing absorption of the gum and alveolus on that side.

The layer of osseous tissue, called cementum, which coats the fangs of the teeth, is liable to become hypertrophied. This may proceed to such an extent that the fang near its extremity may be twice the diameter of the neck. It results from irritation of the dental periosteum, which may itself be occasioned by caries, or necrosis of the tooth. The enlarged fang necessarily compresses and irritates the nerves which pass through the orifice at its extremity to the pulp; and this irritation may be the cause of epileptic seizures, or paroxysms of neuralgic pain. The fangs of the teeth are occasionally absorbed to a greater or less extent, in some rare instances to the same extent that those of the temporary teeth are. The *dental pulp*, a highly sensitive and delicate structure, is very liable to be affected by severe pain from the irritation of caries in the tooth, or even from disease of the pulp of an adjacent tooth, or one situated on the opposite side or even in the other jaw. This is an excellent example of the reflection of sensations. Inflammation often attacks the dental pulp, changing its natural pinkish-gray color to a bright scarlet, and terminating very commonly in its suppuration and death. Sometimes when there exists an opening formed perhaps by caries, into the pulp cavity, the inflammation affects only a part of the pulp. This is intelligible from the circumstance that the secreted matter has a channel of exit, and does not diffuse itself over the rest of the pulp, and also because the irritation of the oral fluids, and of the carious dentine, is confined to the adjacent part of this structure. The inflammation, after having caused the destruction of the pulp, may extend to the dental periosteum, and occasion the death of the fang; it may even extend further to the periosteum of the jaw, and produce necrosis of the bone. The dental pulp may be removed by *absorption* after the cavity of the tooth is laid open by caries, or it may *ulcerate* or perish from *gangrene*. Or again, it may become the seat of a fungoid growth, not of cancerous nature, which is sometimes termed polypus.

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Fig. 209.

Purulent cyst at the fang of a decayed tooth, often the simple origin of most serious mischief.

III.—ABNORMAL CONDITIONS OF THE PHARYNX AND ŒSOPHAGUS.

This part of the alimentary canal may be congenitally absent, or may erminate in a cœcal pouch, or be fused with the trachea, or be dilated nto a sac, or, in rare instances, be traversed by separate fissures. The harynx and œsophagus sometimes become *dilated* throughout, their arietes, and especially the muscular tunic, being hypertrophied. Rokiansky alludes to one case in which the passage was large enough to dmit a man's arm. In some instances, of a less degree of dilatation, he coats are relaxed and attenuated. Partial dilatation appears in the ouches which sometimes are formed by all the coats of the canal, ometimes consist of the mucous membrane only. In the latter case, 'the mucous membrane is protruded between the muscular fibres, and ecomes dilated by the food that enters; it is at last forced out in the hape of a cylindrical appendix, which lies between the vertebral column nd the œsophagus, in a line with the axis of the pharynx, so that all ngesta pass into it, and death from starvation results."

The œsophagus is liable to be *constricted*, either by the compression f external growths, or by cicatrices in its own walls, the results of ormer ulceration or sloughing, or by cancerous formations in its coats. Acute inflammation occasionally attacks the pharynx, or rather its muous lining, chiefly by extension of the disease in cynanche tonsillaris, r simultaneously with the fauces in scarlat. anginos. Sometimes the ffection, though then generally less acute, is independent, and contitutes cynanche pharyngea. Chronic pharyngitis is very common, specially in persons of an atonic habit, or who speak much from the hroat. The mucous membrane appears slightly swollen, and of an ven surface, colored by an uniform redness, and denuded to some ex-

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tent of its investing epithelium. In many cases, the redness is more patchy, and seems to affect more the small veins and adjacent capillaries, and is of a darker tint. M. Chomel has described one form of this affection, in which the mucous follicles are specially the seat of morbid action. He says the arch of the palate is seen to be covered by small red points, which are more thickly disseminated on and near the uvula. These become more numerous and larger as the disease advances, till at length they run into each other, forming ridges and raised patches, between which only a small part of the mucous membrane retains its natural appearance. It occurs much more frequently in males than females, and chiefly between the fifth and seventeenth years. Croupy inflammation sometimes extends to the pharynx and commencement of the œsophagus, and, on the continent at least, diphtheritic exudations are by no means infrequent in this situation. They are essentially similar to those which form on the buccal mucous surface, which have been already described. The pustules of variola are occasionally met with in the pharynx, and those which are caused by large doses of tartar-emetic in the lower third of the œsophagus. The same part of this canal is liable to *softening*, which, usually associated with softening of the stomach, attacks especially the left side, which adjoins the left pleura, and occasions perforation and effusion of the contents of the stomach into this serous cavity. *Fibrous tumors* originating in the submucous or deeper-seated areolar tissues may grow inward, and, obtaining an investment of mucous membrane, hang down into the œsophagus in the form of a polypus. Sometimes they remain without thus protruding in the submucous tissue. Tuberculous deposit is very rarely found in the pharynx or œsophagus. *Cancer* is more frequent in the œsophagus than in the pharynx, in the proportion of thirteen to four; in the former, it mostly affects the upper part, just below the larynx: in both parts, it almost always assumes the form of infiltrated scirrhus, from an ulcerated basis of which soft fungoid growths may afterwards sprout. In the pharynx, according to Dr. Walshe, it generally presents, at least in the early period, "a hard imperfectly circumscribed mass," which may form a tumor visible externally. In the œsophagus it mostly constitutes an annular layer, constricting the canal for a variable distance, and often, by extending outward, producing adhesion of the diseased mass to the spinal column. We have observed one instance in which encephaloid cancer in this situation had proceeded to a considerable extent without producing any symptoms. In the œsophagus of a man who died with peritonitis, there was a thick mass of encephaloid, ulcerated on the surface surrounding the lower part of the

Fig. 210.

Stricture of the œsophagus.

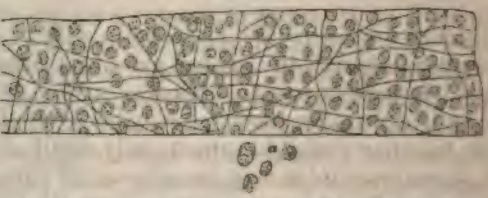
canal, just above the cardiac orifice of the stomach, for an inch and a half. It had evidently originated in the submucous tissue, and grown inward, not contaminating the other coats. Higher up, there were several smaller submucous tumors. Rokitansky mentions the formation of ulcerated openings communicating with the trachea and the bronchi, or even with the aorta and the right pulmonary artery.

IV.—ABNORMAL CONDITIONS OF THE PERITONEUM.

Congenital deficiencies in the peritoneum will, of course, exist when any of the viscera which it invests are absent, or imperfect, or when the walls of the abdominal cavity are in a like state. It appears also that the various folds may of themselves be imperfectly developed, *e. g.* the omentum or mesentery may be unnaturally small or absent. On the other hand, these same folds may be of unusual dimensions, as when a mesentery of more than common length allows the intestines to float up to the surface of an abdomen distended by ascites. Certain pouches are also occasionally met with, chiefly, according to Rokitansky, in the hypogastric, iliac, and inguinal regions, which somewhat resemble commencing hernial sacs, and like them may inclose and incarcerate portions of the intestine. Most remarkable changes in the shape and size of the peritoneum are produced by the dropsical distensions which it undergoes, and the displacements to which it is subjected in various cases of large and inveterate hernia. The peritoneum is extremely liable to *inflammation*, which may be of various degrees of acuteness, or may be chronic *ab initio.* The former is extremely common, and results not only from all causes of irritation applied to it, but also originates spontaneously; or, as Rokitansky avers, in consequence of the rheumatic poison. The inflammation varies much in extent; very often it is general, affecting the whole membrane, but often also it is partial, confining itself to a certain region. The simplest instance of partial peritonitis which we can take is, perhaps, that which occurs when an ulcer is making its way through the walls of the intestine, and threatening to perforate them. Opposite the threatened spot, a patch of injected vessels appears on the serous membrane, which pour out a fibrinous exudation forming a protecting investment, or an adherent medium uniting it to adjacent parts. Inflammation, however, when set up at one part is very prone, as in all serous membranes, to propagate itself to the surrounding, and thus it very commonly happens that peritonitis, which commences in one locality over an inflamed or irritated organ, diffuses itself over the whole membrane till it becomes general. In the early period, injected vessels are very distinctly seen in the inflamed membrane, forming streaks or patches of redness. The injection, however, is seldom very strongly marked, which is, perhaps, due in part to the readiness with which exudation takes place. This is often seen as a delicate thin layer of fibrinous matter closely investing the inflamed surface; sometimes it is so scanty that it is scarcely discernible, unless the surface is carefully scraped, or adjacent intestinal convolutions are separated from each other, when it appears as minute filaments stretching across the interspace. The exu-

dation sometimes collects in the furrows between convolutions of intestines pressed together, and is more manifest there than elsewhere. In instances of sthenic inflammation the exudations are often very abundant, and much puriform is mingled with the fibrinous matter. Serous fluid is also poured out often in considerable abundance, and is rendered turbid by flakes and molecules of fibrin and pus-corpuscles diffused

Fig. 211.



Portion of inflamed peritoneum, with numerous glomeruli between the fibres.

throughout it. In peritonitis, attacking persons who are in an asthenic state the serous and puriform effusions generally predominate. Adhesions are very often found in the peritoneum, connecting the visceral and parietal layers together, and are sometimes of considerable length; in many instances, no doubt, they are the result of partial inflammations giving rise to fibrinous exudation, which is afterwards transformed into areolar tissue; in other cases we are inclined to think the exudation takes place with little or no preceding hyperæmia. The inflamed membrane becomes somewhat thickened by the effusion taking place in its own texture. In the specimen from which the annexed sketch was taken, numerous small corpuscles somewhat resembling glomeruli were seen everywhere among the natural fibres. Bands of adhesion sometimes become the cause of fatal incarceration of the intestine, an opening being formed by these means into which a coil of intestines passes, and after a time having become distended, is strangulated by the abnormal band. A case of this kind is recorded in the Report of the Pathological Society, 1851–52, in which seven or eight inches of the lowest part of the small intestine were strangulated by a ring formed by a strong fibrous band passing from the mesentery to the anterior surface of the rectum. The intestines are very commonly distended by gas in acute peritonitis, which is probably secreted by the mucous membrane under the influence of the irritation to which it is subjected in consequence of the adjoining inflammation. At the same time there is reason to believe that the action of the muscular fibres is more or less interfered with if they are not actually paralyzed, and hence the distension being unopposed is greater than it otherwise would be. Rokitansky says that "hemorrhagic exudation is frequently seen on the peritoneum; it forms large, saturated coagula, disposed in thick layers." Suppuration, as has been said, is not unfrequently a result of acute peritonitis, the purulent matter being, as it were, smeared all over the surface of the membrane; sometimes, however, it takes place in a single part, and forms a circumscribed abscess. On opening the abdomen of a female who had been attacked with peritonitis after the operation of ovariotomy, and who survived several weeks after the inflammation had

been subdued, there was found not only traces of lymph on the surface of the intestines, but a quantity of well-formed pus in the interior of a cavity formed by adjacent convolutions. Had life been prolonged, the pus would have made its way by ulceration into the intestinal tube, and thus been evacuated. Rokitansky, in mentioning this occurrence, speaks of the abscess sometimes discharging itself through the abdominal parietes, or opening this way as well as into the intestine, so that a fistulous communication with the bowel is the result. *Chronic peritonitis* of a simple kind is not of common occurrence; a case is, however, recorded by Andral, in which serum, turbid with albuminous flocculi, was found in the serous cavity after death, while ascitic effusion had existed for more than the last month of life, unattended with pain or any evident symptom of inflammatory action. We think we have seen a case somewhat similar. There is, however, another form of what may, perhaps, be termed chronic peritonitis, though we doubt very much its essential dependence on any inflammatory process. In this, the serous surface is invested closely for a greater or less extent by a firm, whitish, false membrane, which can be pretty easily detached from the subjacent peritoneum, and appears, when held up to the light, of much thinner texture in some spots than in others. The situation in which the false membrane is most completely formed is upon the surface of the liver, to which it forms sometimes a complete capsule, compressing and atrophying it, and giving rise to ascites from interference with the free passage of the blood through the structure of the gland. We incline to the belief that these exudations, which we have seen on the surface of the pleura, as well as on that of the peritoneum, are the results of an abnormal condition of the fibrinous constituent of the blood, in consequence of which it is prone to be effused either in the substance of tissues, as in cirrhosis, and puckering of the cardiac valves, &c., or on the surface, as in the case before us. One of the best-marked varieties of chronic peritonitis is that which is often justly called "tubercular," from its being essentially dependent on the presence of tubercles in the peritoneum. These appear sometimes as semi-transparent gray granulations, sometimes are more opaque, though still of miliary dimensions. They are diffused everywhere throughout the subserous tissue, but are said by Dr. West to be most numerous on the surface of the diaphragm, or on the abdominal walls in the neighborhood of the spleen, while the parietal peritoneum is not so much affected as other parts. The chief tubercular deposit is sometimes in the omentum, and may assume the form of crude tubercular masses. Inflammatory irritation is produced by the tubercles acting as foreign bodies; exudation of lymph takes place, and adhesions are formed between adjacent parts of the serous surface, which are often so close and dense that the intestines or other viscera are torn in making an attempt to separate them from each other. We have seen the whole of the serous sac in this way entirely obliterated. There is usually some serum in the peritoneal cavity, but no puriform matter, unless, as occasionally happens, acute inflammation has supervened upon the chronic, and proved fatal. The tuberculous deposit sometimes undergoes softening, as in other situations, and the extension of this process may cause perforation of the walls of the in-

testines, and either establish unnatural communications between distant parts which have become adherent together, or lead to the effusion of the intestinal contents in the serous cavity. The latter event, however, is more likely to be produced by the softening of tubercle in the submucous tissue, which is often present there at the same time as well as in the mesenteric glands. There is no relation apparently between the amount of deposit in the peritoneum, and in these two other localities. The lungs and bronchial glands are often tuberculous when the peritoneum is affected, but often in a much less degree, and they may sometimes be exempt altogether. Rokitansky describes the muscular tunic of the intestines as being still more affected in this disease than in acute peritonitis; it becomes pale, is easily lacerated and broken up. This gives a further reason why laceration often occurs on attempting to separate the intestines which are matted together.

The peritoneum often appears somewhat thickened, of a dull, dense, whitish sodden aspect in cases of chronic ascites. We have examined the membrane thus altered, but were unable to find any very marked alteration in texture, only that the tissue seemed more granular and less purely fibrous than natural. A local change of somewhat the same kind is often seen in the peritoneal covering of the liver and the spleen. In these it forms dense whity patches, which shade off gradually at their margins, and are for the most part quite free from bands of adhesion on the surface. The change in the splenic capsule is often so great that it has been termed "cartilaginification." When the exudation, which in all these cases takes place into the subserous tissue, contracts and draws the part together, it produces "lobulated laminæ, and projecting granulations," of a firm, dense structure. Calcareous matter is sometimes deposited in these fibrous formations, and gives rise to "compact, smooth, or uneven lobulated plates of varying thickness." It appears from Rokitansky's account, that false membranes, the result of inflammation, may be so disposed as to form serous cysts, which obtain an internal smooth lining, and are either pedunculated or sessile on the peritoneum.

Cancer attacks the peritoneum in some very rare cases primarily, but most often by an adjoining growth extending to it, "perforating it, and penetrating into its cavity." The disease is sometimes of the encephaloid variety, but more often of the colloid. The latter, when the process of development is acute, is often spread over the entire serous surface, in the form of small, miliary nodules, in some parts clustered together. "Sometimes it occurs as a layer of areolar cancerous tissue, varying in thickness, or as a circumscribed, round, lobulated aggregation. The omentum is very commonly found to shrivel up and to degenerate into a transverse band; or, in the opposite case, with an enormous increase of size into areolar cancer." Cancerous growths not uncommonly originate in the *post-peritoneal cellular tissue* just in front of the spine. In this part they are firmly adherent to the vertebræ: are of homogeneous (probably firm scirrhous) texture, and confounded with the crura of the diaphragm. In its peripheral parts the growth has a more loose and lobulated structure. Dr. Walshe, tracing the progress of the mass from its point of origin, says, "it spreads upwards,

extends to the stomach, presses under the liver, penetrates between the laminæ of the transverse mesocolon, twists round the duodenum and pancreas, and, pushing forward the stomach (with the small curvature of which it contracts adhesion), forms a tumor in the epigastrium. Such tumors mould themselves upon these various parts and organs in so close a manner, that, after separation, the surface of the mass retains the impressions of the adjoining viscera." The course of these growths, though rapid, is often for a long period unattended with pain or disturbance of the system.

V.—ABNORMAL CONDITIONS OF THE STOMACH.

In very imperfect monstrosities, especially the acephalous, the stomach is either wanting, or very imperfectly developed. It is also absent occasionally, according to Rokitansky, in individuals otherwise normally built, and provided with a well-developed intestinal tube, or it may be only indicated by a small saccular dilatation of the œsophagus. The *shape* of the stomach is sometimes found remarkably altered; its cavity being partially divided into a cardiac and pyloric portion by an annular contraction, or even still further subdivided into three or four sacculi, so as to present some resemblance to the multiple stomachs of ruminants. These peculiarities of shape may either result from congenital malformation, or at least, in their minor degrees, from irregular contractions of the muscular coat, or from destruction of substance and subsequent cicatrization. Sometimes the stomach deviates from its usual shape, in the way of assuming greater simplicity; it is destitute of its cardiac cul-de-sac, and the œsophageal opening is quite at its left extremity. The stomach is liable to great variations of *size;* these within certain limits are physiological, and are manifestly contemplated in the peculiar convoluted disposition of its mucous lining. A healthy stomach, when empty, naturally contracts upon itself, and this to such an extent that its cavity is wellnigh obliterated. This is purely the effect of the unopposed action of its muscular coat, and is no evidence of disease in the viscus. It may proceed from starvation, or from stricture of the œsophagus. Contraction of the cavity of the stomach in a less degree may be produced by hypertrophy, and cancerous disease of the coats, or by the cicatrization of extensive ulcers. *Dilatation* of the stomach, often to a considerable extent, is of very common occurrence, and depends, partly, on copious secretion of gas from its lining membrane, and partly on loss of contractile power in its muscular fibres. In its more extreme degrees, it is generally the result of obstructive disease at the pyloric outlet, in consequence of which the ingesta accumulate within the cavity. The distension in such cases is sometimes so enormous that the stomach extends over the entire abdominal cavity. Rokitansky says, "that repeated repletion, in consequence of a morbid appetite," may become the cause of as great distension as when the pylorus is obstructed; or that this may also "occur as a result of paralysis from concussion, traction, or dislocation, produced by large scrotal herniæ, and that it kills slowly with vomiting, with or without gangrene of the

mucous membrane, under symptoms of complete paralysis." The coats of the stomach may be abnormally thick, either in consequence of cancerous disease, or from simple hypertrophy of the muscular layer. The pyloric outlet is generally the part where muscular hypertrophy shows itself, and here it seems to affect more especially the layer of annular fibres. *Atrophy* of the coats, speaking generally, is most commonly observed in cases attended with much emaciation; it occurs also sometimes in consequence of extreme dilatation of the cavity, sometimes spontaneously. The muscular layer is, in most cases, the one most palpably affected; the mucous membrane is, however, not uncommonly atrophied also, as we shall more particularly describe when speaking of textural changes. The following list of abnormal situations, which the stomach may occupy, is given by Rokitansky: "It may lie external to the abdominal cavity in eventration, and in umbilical hernia; in the left side of the thorax, the diaphragm being wholly or partially absent on that side;" it may lie vertically, as in the fœtal state; or with the fundus on the right ride, as in general lateral transposition. The foregoing are congenital malpositions; the following acquired. The stomach may protrude externally after extensive wounds, or make its way into the thorax, after injuries to, or ruptures of, the diaphragm; it may be carried down into large hernial sacs, especially umbilical and scrotal; or be displaced by new growths or enlargement of adjacent organs; or sink lower itself, in consequence of increase in size, as in the case of a scirrhous pylorus.

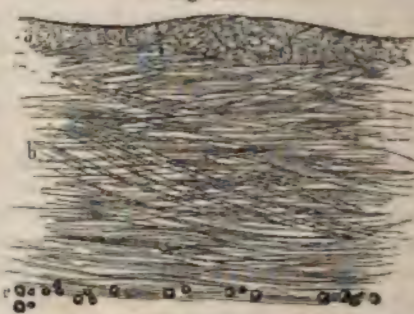
Acute inflammation of the stomach but rarely, if ever, occurs, except as the result of irritants directly applied to it. The cases related by Andral give proof, however, of its existence as an idiopathic affection, or as the sequel of rheumatism, or of epidemic cholera. We quote the account he gives of the morbid changes. In Case 1, the stomach was strongly contracted so as to be nearly the size of the transverse colon: "Its inner surface, over nearly its entire extent, was of a brownish red. This color had its exclusive seat in the mucous membrane, which had become in every part very thick, and was at the same time very friable. On its free surface there was discovered a multitude of small red or blackish points, which seemed to have their principal seat in the villi; however, beneath these the body of the mucous membrane was red, and, as it were, penetrated with blood; in no part could this membrane be detached, it gave way under the forceps, and in several points it resembled a pulp without any consistence." Near the pylorus, the mucous membrane resumed its normal consistence, and was of a grayish color. In Case 2, also, the stomach was strongly contracted. Its mucous membrane was of a dark red color, over the entire surface of the great cul-de-sac, and over all the posterior surface from the cardia to the pylorus. This redness penetrated the entire substance of the membrane, which had lost its consistence in every part where it was red; in some points it was merely a sort of pulp. Towards the anterior surface the mucous membrane presented a slate-colored tint, without its consistence being much changed; near the pylorus some mammillation was observed. The surface was covered by a viscid thready mucus. In another case, also idiopathic, the parietes of the stomach were remarkably softened,

"over all the left portion of this viscus, its tunics, from the peritoneal to the mucous, had no longer any consistence; they gave way under the fingers as a sort of pulp. Wherever this softening existed, the parietes of the stomach were of a dark red color, and as it were ecchymosed. Near the pylorus the parietes of the stomach resumed their natural consistence," and of a grayish tint. In persons dying of gastritis, consecutive to a malignant cholera, Andral found, at an early period of the disease, the mucous membrane red and softened; at a later period it was sometimes in the same condition, sometimes brown or slate-colored, and its tissue thickened and indurated. Judging from our examinations of other inflamed mucous membranes, we feel no doubt that the microscope would show, in such instances, more or less hyperæmia of the vessels, with abundant granular exudation, stained by exuded hæmatin in the substance of the mucous tissue, as well as loss of its investing epithelium, and wasting or breaking up of its glandular tubuli. We feel the more confidence in Andral's description above quoted, because he has so carefully distinguished inflammatory hyperæmia from mere passive or post-mortem congestion, or red staining, and has contended so prudently against the extravagances of over-zealous Broussaians. We have recently examined an excellent instance of intense hyperæmia of the stomach. The whole mucous surface was of a deep red, almost black; the subjacent tissues were much less affected. The cavity was empty, and the organ was much contracted. The surface was uncovered by mucus, only a little alkaline fluid lay in the furrows between the rugæ. The capillaries were gorged with blood in every part of the membrane, they were seen running parallel to the tubes in their whole length, but those which adjoined the free surface (which are always the most congested), had given way in numerous spots, and saturated the tissue round them with extravasated blood. The tubes were healthy, and there was no apparent exudation among them. The patient died with cardiac hypertrophy and general dropsy. The liver and spleen were much congested, and it is pretty certain that the hyperæmia of the stomach was passive rather than active.

As Rokitansky observes, we have rarely, if ever, the opportunity of observing the first stage of acute *catarrhal inflammation* of the stomach, but we may reasonably infer that it consists, as in other parts, of a more or less considerable hyperæmia, which relieves itself by a copious exudation of mucus upon the surface, instead of a fibrinous exudation in the substance of the tissue. A chronic catarrhal state is by no means uncommonly met with, the anatomical characters of which Rokitansky enumerates "as a dark reddish-brown, or slate-gray, or even blackish-blue discoloration of the mucous membrane, copious secretion of a stone-colored, occasionally glassy pituita, thickening, increased condensation and induration, *i. e.* hypertrophy of the mucous membrane, which presents itself in various degrees: (α.) In the lowest degree, the mucous membrane shows simply an increase of thickness and hardness in its tissue; (β.) In a higher degree it presents, in addition to its increased thickness, an uneven, racemose, or warty surface, a surface mamellonée; (γ.) In a still more advanced degree, it forms prolongations in the shape of permanent firm folds or of polypus. The submucous cellular tissue,

and the muscular coat, also participate in this hypertrophy in various degrees—the entire parietes of the stomach presenting unusual thickness, firmness, and hardness. The pyloric portion is the chief seat of chronic catarrh, and it is there that hypertrophy of the mucous and other membranes is most prominent." Andral remarks that, in chronic gastritis, the mucous membrane may appear after death to be in a perfectly natural state, or at least to have undergone no alteration discernible by the eye. The subjacent tissues, and particularly the submucous areolar tissue, are, however, in these cases more or less affected. In the majority of instances, however, the color, the consistence, and the substance of the membrane are variously changed. A gray slate, brown, and more or less deep black tint are often observed, as well as sometimes a dull white milky aspect. In regard of consistence, the mucous membrane may be indurated, or softened; the latter is more frequent than the former. Induration may exist with all the different shades of color just mentioned. With respect to its substance the mucous membrane may be thickened or attenuated, or may remain unaltered. A

Fig. 212.



Vertical section of mucous membrane of stomach, the tubes being completely wasted and replaced by fibroid tissue. (*a*.) Remains of mucous membrane. (*b*.) Fibroid tissue. (*c*.) Fat-cells.

state of thickening may coexist with induration or with softening; the former combination is peculiar to chronic gastritis, and affords a good example of false hypertrophy. Induration, like the thickening, may be partial or general. Attenuation of the gastric mucous membrane is most often met with towards the great cul-de-sac, in the same situation where softening is most frequent. Andral says: "Sometimes, however, I have found the mucous membrane towards the pylorus, so attenuated that it resembled a sort of transparent extremely fine web. On attempting to raise it, it was changed into a reddish-white pulp, as happens in certain degrees of softening." He admits, what we shall presently remark, that this attenuation may occur as a pure atrophy totally independent of inflammation.

We have lately examined carefully with the microscope more than a hundred stomachs taken indifferently, and have published, in the *Assoc. Journal* for Oct. 7th and Jan. 27th, 1854, the details of the following varieties of change: (1.) One of the commonest, especially in its minor degrees, consists in the infiltration of a low fibroid tissue loaded with

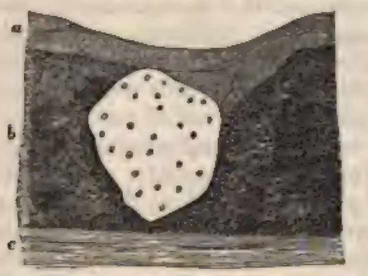
nuclei among the tubes, which themselves undergo atrophy, so that at last the mucous membrane totally loses its tubular aspect, and becomes a mere fibroid stratum, more or less densely set with nuclei throughout. In this state the basement-membrane may still persist, and the thick-

Fig. 213.

Vertical section of mucous membrane of stomach, showing the lower parts of the tubes, and a nuclear mass extending among them upwards. (*a.*) Tubes. (*b.*) Nuclear mass. (*c.*) Submucous tissue.

ness of the tissue be little diminished. In some instances the nuclei disappear, and the fibroid stratum develops fibres more decidedly. (2.) There are formed masses of nuclear particles, most often at the bases of

Fig. 214.



Cavity formed in the mucous membrane of stomach, by the disintegration of a nuclear mass. The surrounding tissue is pervaded by nuclear deposit. (*a.*) Basement-membrane of surface. (*b.*) Altered mucous membrane. (*c.*) Submucous tissue.

the tubes encroaching upon them, often also in the substance of the mucous membrane, and sometimes at its surface: these are sometimes circumscribed, sometimes diffused, and then pass into the preceding forms by gradual shades. (3.) The nuclear deposits sometimes seem to give rise to cystic cavities, or these may form from dilatations of the tubes, or arise *de novo*, as in other situations. (4.) The mammillated condition appears to depend on a process of local atrophy, at least in most cases, the tubes being wasted in the track of the furrows, which

sometimes are so deep as to fissure the membrane down to its corium. (5.) A fatty state is very commonly met with, and in two forms, one in which the epithelium is bulky and the tissue healthy, or nearly so; the other, where the epithelium is atrophied, in consequence, generally, of pressure by new-formed fibroid tissue upon the tubes. (6.) The tubes in the pyloric region are often found changed in the following manner: the continuous row of tubes is interrupted, and there are seen at intervals instead groups of convolutions containing a fatty wasted epithelium, and not possessing any manifest outlet on the surface. We think that inflammation is not the most essential moment in these changes. The nuclear masses, when not of large size, may be regarded as identical with the naturally existing solitary glands.

Croupy inflammation resulting in fibrinous exudation, which forms a false membrane, sometimes of regular areolar surface, is very rarely seen at least in England, and is said by Rokitansky to be a "sequela or degeneration of exanthematic processes, as of variola, typhus, pyæmia, and particularly puerperal phlebitis. Sometimes inflammation of a low erysipelatous kind attacks the *submucous cellular tissue* of the stomach, and occasions suppuration. The pus, after a time, escapes by numerous irregular cribriform openings into the cavity of the viscus.

We proceed to notice the *effects of caustic fluids*, such as the mineral acids, which have been swallowed. The mucus in the mouth and fauces is coagulated into flocculent masses, the epithelium is detached here and there, and "converted into a thick grayish-white, rugose layer," and the subjacent mucous membrane is pale. If the caustic fluid has penetrated more deeply, "the superficial layers of the mucous membrane of the fauces and œsophagus are found congested, of a dirty, whitish, leaden hue, and the capillary network blackened by its carbonized contents. The lower strata of the mucous membrane, and the submucous cellular tissue, present serous infiltration. In the follicles at the root of the tongue, the mucous secretion is coagulated into dirty white masses. In a still higher degree of corrosive action, the entire mucous membrane is destroyed, and converted into a dirty gray mass, which is traversed by black vessels; the submucous cellular tissue is infiltrated, and partially ecchymosed; the muscular coat of the œsophagus itself is shrivelled, pale, ashy. In the highest degree, the mucous membrane of the œsophagus, together with the submucous cellular tissue, is converted into a soft, black mass, which is distended by a sanguinolent fluid, and is easily detached from the muscular coat. The latter is itself either destroyed in the same manner, or is perfectly colorless, friable, and presents an ashy, gelatinous appearance. The mucous membrane of the stomach," in consequence of being longer in contact with the corrosive substance, "almost invariably suffers the changes of the last degree but one, though in varying extent and thickness. It is either affected in single folds, or streaks which pass from the cardiac orifice to the lesser curvature, and from the large curvature to the pylorus; or over a large extent; or we find the entire surface converted into a black carbonaceous mass, of several lines in thickness, distended by sanguinolent fluid, and consequently presenting a tumefied appearance." The muscular coat is affected, and the parietes of the stomach are often perforated.

"The acid affects the neighboring organs through the membranes, and thus either coagulates or tans the contained fluids, fuses the tissues into a gelatinous mass, or carbonifies them; the discoloration produced is always very marked. In many cases, not only the blood of the neighboring bloodvessels, but also of the larger trunks, and even of the aorta, is changed into pultaceous, pitchy, greasy, black cylinders. Beyond the stomach, and especially in the duodenum, and at the commencement of the jejunum, the effect of the lowest degree is exhibited in coagulation of the intestinal mucus, and of the chyle, in corrugation and opacity of the epithelium, in the tanned state of the mucous membrane, and the dark injection of its vessels." The highest degrees of corrosive action are rapidly fatal, "the lowest degrees are followed by exudative inflammation, the mortified epithelium sloughs, and being replaced by a new formation, as soon as the reaction has abated, recovery ensues." In all the higher degrees, inflammation, passing into suppuration, produces the separation of the superficial mortified layers. The suppurating process may be protracted, or may terminate early with the formation of cicatrices. "According to the depth to which the tissues are destroyed, the loss of substance is repaired under a formation of structures that vary in size and consistency." When the mortification is limited by the submucous cellular tissue the latter becomes condensed, and "forms, at some places, projecting ridges, or valvular, and even annular, duplications towards the œsophagus;" in this way peculiar membranous strictures are produced. "If the muscular coat itself is involved, it is partially or entirely destroyed, and the walls of the œsophagus are converted into a fibro-cellular firm tissue, which contracts, and thus produces the most important and most resisting strictures." Chronic suppuration sometimes occurs as the result of profound injury, leading to the formation of abscesses and sinuses of the muscular coat, and of the surrounding cellular sheath of the œsophagus. These may produce perforation of adjoining passages, the trachea or bronchi, or may heal, leaving considerable contractions of the tissues and strictures. Cicatrices and strictures are formed in the same way, though less frequently, in the membranes of the stomach. The morbid changes produced by arsenic are as follows: "At one or more points to which the powder happens to attach itself to a larger amount, the mucous membrane appears plicated and tumefied, reddened, invested by a detached epithelium, and a tawny exudation; its tissue is softened, pultaceous; and at the spot where the white grains of arsenic are attached, it is converted into a yellowish or greenish-brown slough." The tissue intervening between these solitary foci is often quite healthy.

Ulceration of the coats of the stomach is much less frequent than that of other portions of the intestinal canal. It occurs sometimes as the result of chronic gastritis, or of the corrosive action of poisons. These ulcers require no particular notice; but there is one particular kind which is rather peculiar to this organ, and which is of especial interest, from its occurring in tissues which otherwise appear quite healthy, and from the serious and rapidly fatal effects to which it too often gives rise. Rokitansky terms this the perforating gastric ulcer, on account of its having a decidedly marked tendency to perforate the parietes of

the stomach. He describes it as follows: "In a well-defined case there is, in the region of the pylorus, a circular orifice of from three to six lines in diameter, with a sharp peritoneal edge, as if a round piece of the gastric parietes had been punched out. When viewed from within, the loss of substance on the internal membranes of the stomach, and

Fig. 215.

Perforating ulcer of stomach; the mucous membrane is puckered into folds round it.

especially of the mucous layer, appears more considerable, so that the edges of the hole seem bevelled off from within outwards." In some cases the margins of the ulcer are quite smooth and thin, in others thickened and indurated. "The pyloric half of the stomach," Rokitansky proceeds, "is the seat of the ulcer: it is most frequently found in the middle zone of this portion; it is oftener seen at the posterior than at the anterior surface, almost always near to, and frequently at, the lesser curvature; and it occurs in extremely rare cases only at the fundus." A similar ulcer may form in the upper oblique portion of the duodenum, but not, as far as observation has yet shown, in any other part of the intestinal canal. The size of these ulcers, Rokitansky testifies, may equal that of a cheese-plate; we have never seen them exceeding that of a half-crown. Their form is commonly circular, at least in the outset, though they often become elliptical or quite irregular, as they extend. Sometimes the ulcer enlarges in its transverse diameters, so as to obtain a zonular form. Sometimes two ulcers coalesce together more or less completely. "In the majority of cases there is only a single ulcer," (there is no mention of more than a single one in three cases which are recorded consecutively in the Report of the Pathological Society for 1847–48,) "but frequently there are two or three, occasionally four or five, and these are then commonly placed above or near to one another at the posterior surface of the stomach, or at the lesser curvature." When the ulcer is perfectly circular, the narrowing of its area, as it extends in depth, is very marked, the muscular coat is less extensively destroyed than the mucous, and the peritoneum again less extensively than the muscular; the perforation, in fact, taking place, as Rokitansky describes it, in the centre of the included circle. The exact nature of the process by which the ulceration commences, is not at all ascertained, at least, has not yet been the subject of direct obser-

vation. Rokitansky writes: "It is probable that it commences with an acute, circumscribed, red softening (hemorrhagic erosion); or with a circumscribed sloughing of the mucous membrane; it is still more probable that the ulcer increases in this manner, the tissues at the base of the ulcer sloughing and exfoliating, layer by layer." He thinks that "the process offers a valuable analogy to sloughing of the lungs." We think the gastric ulcer may be very properly compared to the simple ulceration of the cornea, which it resembles closely in several respects. Both, when the system has made no reparative effort, may have smooth level margins. Both may heal by the deposition of fibrin at their base and around their margins. Both show, when they advance unchecked, a decided tendency to perforate the tissue in which they exist. In both the ulceration is evidently not the result of violent inflammation, but of a local loss of substance or disintegration. It seems as if the tissue slowly liquefied, molecule by molecule, in a given part, in consequence of defect of assimilative power. The process in the case of the cornea is evidently not identical with sloughing, such as occurs in purulent ophthalmia, and there is reason to believe that the same is true also of the analogous change in the gastric parietes. Rokitansky states that the ulceration "is invariably accompanied by chronic catarrh and blennorrhœa of the gastric mucous membrane." This we much doubt, for, though pain and various dyspeptic symptoms are complained of in most cases where ulcers of this kind are found, yet this is not constant, and certainly the amount of the dyspepsia is no indication whatever of the existence of ulceration. The point we wish especially to notice is, that the ulcerative action is not in any way dependent on irritation or inflammation, but on a loss of vital assimilative power in the part affected. The bearing of this part upon the treatment is most important. Dr. Copland states, and some of our own observations are confirmatory, "that this affection is most frequent in needlewomen, or female servants. The patients, in most instances, have been anæmic, or suffering from disordered menstruation, as well as from pains in the stomach, but "have generally been able to pursue their avocations, and to take their food, up even to the period of the fatal seizure." The ulcer, especially when seated near the smaller curvature, is apt to involve some arterial branch, from which blood is poured out in abundance. The hemorrhage may be so copious as to destroy life at once, but more frequently death does not occur till after repeated attacks. The deeper the ulcer has extended, the larger in general are the vessels it meets with, and the more serious, in consequence, the loss of blood. It has happened, that an ulcer, after perforating the coats of the stomach, has lighted upon the pancreatic duct, and produced a fistulous opening into it. The most dangerous situation for an ulcer to occupy is the lower half or two-thirds of the anterior surface of the stomach, as, in case of perforation, there is no organ to which it can easily become adherent. On the posterior surface, adhesions form between the stomach and pancreas, or the adjoining lymphatic glands, and on the upper and pyloric part of the anterior surface the escape of the gastric contents into the peritoneal cavity is sometimes prevented by the left lobe of the liver. In a remarkable case, recorded in the Report of the Pathological Society,

1847–48, p. 252, the barrier opposed to the extension of an ulcer by the left lobe of the liver proved insufficient, as the destructive process continued until it perforated the diaphragm, and gave rise to hepatization and a gangrenous cavity in the lower lobe of the left lung. These ulcers may heal at any period of their course; it is not uncommon to find the cicatrices, which are their results, on the inner surface of the stomach. There can be no doubt that an effusion of plasma undergoing development into fibroid tissue, is the means whereby the separation is effected. This takes place both in the margins of the ulcer, producing thickening and subsequent contraction, and also at the base, which it lines with a thin smooth layer. Cicatrices of this kind present a depression of the size of the ulcer, surrounded by thickened and elevated margins, others, where more contraction has taken place, are of a linear or corded shape.

Hemorrhagic erosion of the gastric mucous membrane is thus described by Rokitansky: "There are round or roundish spots of the size of a pin's head or pea, or narrow elongated streaks, at which the mucous membrane appears dark red, lax, soft, bleeding, and presenting a depression in consequence of loss of substance or slight erosion. Sometimes this loss of substance involves the entire thickness of the mucous membrane and the submucous cellular tissue, and produces an appearance of small round, or striated ulcers. This process is invariably accompanied by hemorrhage," the effused blood being mixed in a more or less altered state with gastric mucus, which is poured out by the membrane affected with recent or inveterate catarrhal inflammation. The erosions are often very numerous, studding, perhaps, every part of the stomach, with the exception of the fundus; their chief seat is at the pyloric portion. They are not peculiar to any form of disease. Microscopic examination of one of these ulcers showed the surface sunk in, the basement-membrane gone, and the tubes quite atrophied and replaced by low fibroid substance, infiltrated with diffused yellow pigment.

Softening of the stomach requires an especial notice, as an affection of great importance, though as yet very imperfectly understood. One form of it, called by Cruveilhier *gelatiniform softening*, occurs, especially in infants, between the age of four and eighteen months. The process commences with the mucous membrane of the fundus, and "extends to the muscular coat and the peritoneum, converting them and the intervening cellular tissue into a grayish or grayish-red, transparent jelly, with a yellowish tinge, through which single dark-brown streaks, the broken-down bloodvessels, are observed to pass. The softened portion of the stomach tears at the slightest touch," and rents in it take place, sometimes perhaps during life, but more frequently after death. The softening process sometimes extends to the diaphragm, causing perforation of it, and effusion of the gastric contents into the left pleura. Dr. Copland expresses his opinion, that "the softening often exists to a considerable degree previously to death; but the advanced stage of disorganization, and more especially erosion and perforation are early consequences of dissolution, which the fluid of the stomach may have been, more or less, concerned in producing." The same authority states that the disease is almost endemic in certain places, and epidemic in

some seasons. "It may appear in the course of infantile remittent fevers, of hydrocephalus, or of chronic bronchitis; or it may follow the cholera infantum, or scarlet fever, or diarrhœa, especially after weaning, or when the infant has not enjoyed the advantage of a healthy nurse, or is brought up by hand." "General anæmia," as Rokitansky testifies, "which is particularly apparent throughout the intestinal canal, and general collapse and wasting, which are chiefly evident in the muscular tissue, are constant accompaniments of this disease." It has been much debated whether the affection should be regarded as of an inflammatory nature; but the entire absence of hyperæmia, or of the products of inflammation in the softened tissue, must be considered as almost decisive of the question. Rokitansky observing its frequent supervention on hydrocephalus, or some cerebral affection, suggests that "the proximate cause may be looked for in diseased innervation of the stomach, owing to a morbid condition of the vagus, and to extreme acidification of the gastric juice." The gastric mucous membrane is not unfrequently found softened in adults who have died from various diseases. Andral notices its occurrence in many chronic diseases, especially pulmonary; and also as the first sign of failing power in old persons, whose health has been generally good. The morbid change betrays itself by anorexia, uneasiness and weight at the epigastrium; emaciation and rapid loss of strength. Death takes place in the way of asthenia, without prominent affection of any organ, and the autopsy only reveals gastric softening, with or without hyperæmic injection. The color of the softened tissue, according to Andral (and we are quite disposed to agree with him), is by no means uniform; it may be normal or paler than natural, or of a dead white, or red or brown. Rokitansky, on the other hand, says "the parietes of the stomach are converted into a more or less saturated dark brown or reddish pulp." In our examinations, we have very commonly, indeed, found the splenic region of the stomach more or less softened. The mucous membrane appears thinned, more translucent than usual, and dark stained; under the microscope, its tubes appear in various degrees of disintegration. The change is, we think, partly post mortem, partly owing to failing nutrition; its constant seat in the splenic region seems to be connected with the greater thinness of the walls, especially the muscular, in that part, in consequence of which it is very often abnormally distended. In some cases the other coats are involved, as well as the mucous membrane, and even the diaphragm may be thus perforated. "The stomach is found to contain large quantities of a fluid resembling coffee-grounds or ink, which is often vomited during life." The œsophagus, as we have before mentioned, is sometimes contemporaneously affected in its lower third, in consequence of which, perforations and effusion into the left side of the thorax may result. "The fundus is the seat of all the softening processes of the stomach," from whence they extend to the larger curvature. This applies not only to the softenings which commence during life, but, at least in the majority of cases, to that which occurs only after death. It is often difficult, as Rokitansky allows, to distinguish certainly between the mere cadaveric chemical changes, and those which take place from alterations in the vital nutritive processes. The distinction, however, may generally be made by

attending to the following circumstances, which he enumerates: (a) "the absence of all symptoms during life which indicated softening, or the morbid processes that give rise to it; (b) sudden death, from natural or other causes, during the digestive act, while the stomach is filled with chyme, without previous illness; (c) limitation of the softening to the mucous membranes, and especially to the projecting folds, so as to form streaks; (d) and at the same time, its extension beyond the ordinary boundaries of morbid softening—its development being most remarkable at those points at which there is a stagnation of the greatest quantity of the gastric contents;" this latter circumstance determines the seat of post-mortem softening to be the part which is most depending. An experiment performed by M. Cameron, illustrates very well the influence of impaired vitality in promoting the softening of the gastric tissues. A fluid obtained from the stomachs of two children, who died from gelatiniform softening, was introduced into the stomach of a living rabbit, and produced no injurious effect; the viscus being found quite healthy when the animal was killed. Another rabbit was treated in the same way, having previously had its pneumogastric nerves divided, the mucous membrane of the stomach was found in a state of softening. If the nerves only were divided, no softening took place. This experiment seems confirmative of the opinion of Dr. Copland, noticed above.

Fatty tumors, originating in the submucous tissue, and increasing in size, may either press inwards towards the cavity of the stomach, or outwards, towards the peritoneal sac. In either situation they may be sessile or pedunculated. *Fibroid nodules* sometimes form in the areolar submucous tissue, "chiefly in the vicinity of the cardiac orifice, and the lesser curvature." *Erectile tissue* may be developed at the free extremity of polypoid growths, or may occupy a larger surface of a sessile tumor. *Tubercle* is very rarely seen in the stomach, and only occurs in cases "where intestinal tuberculosis has advanced to an extreme degree." *Cancerous disease* of the stomach is frequently met with; this organ ranks next to the uterus in the list of mortality from this cause. Primary cancer exists in the majority of cases. Dr. Walshe speaks of secondary "as almost unknown," except where it invades the organ from extension of adjacent growths. It is not uncommon to find a solitary growth in the stomach, no other part being implicated, as in a case we have recently examined. "The pylorus," says Rokitansky, "indifferently at all parts of its circumference, is known to be the chief seat of primary fibrous and areolar cancer of the stomach. From this point the degeneration extends chiefly along the lesser curvature over the pyloric half of the stomach; in many, though severe cases, it affects the entire stomach, attacking the fundus last, which, however, generally remains partially free. The parietes of the stomach may attain an inch in thickness, being rigid and generally tuberculated on their inner surface; the cavity of the stomach will at the same time be diminished in size." Dr. Walshe states that cancer of the orifices may extend to the duodenum or the œsophagus. Rokitansky affirms "that cancer of the pylorus is accurately bounded by the pyloric ring, and never extends to the duodenum," whereas, cancer at the cardia invariably involves a portion of the œsophagus. We certainly think that scirrhous disease

of the pylorus does not extend much beyond its original site, at least along the intestine, although it may propagate itself to the head of the pancreas, or the adjacent lymphatic glands. Commonly, as Rokitansky describes it, the scirrhous pylorus is bound down by the degeneration of the tissues lying behind it; but, in other cases, it remains movable, and may be felt as a distinct tumor having descended more or less over to the lower part of the abdomen. The pylorus, the cardiac orifice, the greater, and, lastly, the lesser curvature are liable, according to the order in which we have placed them, to be the seat of cancer. Fibrous cancer undoubtedly is the most common, *i. e.* scirrhus, or, as we are inclined to think, a combination of scirrhus with colloid. Medullary cancer ranks next, according to Rokitansky, and areolar or colloid last.

Fig. 216.

Scirrhus Pylori. At the diseased part, the walls of the stomach are extremely thickened, and of a whity color.

He notices the frequent primary combination, and the yet more frequent secondary, of scirrhus with encephaloid, or of both with colloid. The following description was taken from an exceedingly well-marked specimen of scirrhus pylori, in which the walls of the passage were so thickened as to be nearly an inch in diameter. The cut surface presented a whity grayish tissue, contrasting well with the injected mucous membrane, and exhibiting a distinct striation vertical to the axis of the canal. A section under the microscope showed grayish-white bands, separated here and there by transparent gelatinous matter. The bands consisted of homogeneous, faintly-mottled substance, occasionally divisible into fibres closely resembling those of organic muscle, and, like them, exhibiting elongated nuclei when treated with acetic acid. Towards the mucous membrane this close stroma was replaced by a loose fibroid tissue, forming circular loculi of various sizes, which were filled with very various forms of cell-growth. Among them granule-cells were often apparent, but the main mass consisted of nuclei and low developments of them. Some large mother-cells were seen, containing several well-formed nuclei and granulous matter: in the interspaces between the fibrous bands these mother-cells had attained a gigantic size, and appeared to constitute the loculi; one of them was distinctly bifurcated at its narrow end, and the branches were of some length. Dr. Bennett, v. p. 43 of his work, doubts the cell character of these conglomerate

masses, chiefly from the absence of a cell-wall. We are, however, inclined still to believe that the loculi, in this instance, and in colloid generally, resulted from the development of an endogenous growth within parent cells. In this case, we consider that there was a combination of colloid with scirrhus, the former being constituted by the colloid substance. In Dr. Bennett's xxi. *Observation*, the alteration which had taken place in "the walls of the stomach was wholly of a fibrous character." No cancer-cells were detected, only elongated and fusiform nuclei; but they were numerous in the enlarged mesenteric and lumbar glands. This latter circumstance is, we think, decisive of the truly cancerous nature of the morbid change in the stomach. Such a case may then be regarded as one of pure scirrhus, upon which encephaloid growths are sometimes secondarily developed, appearing as fungus or cock's-comb-like bleeding excrescences. Encephaloid, however, either in the form of knotted tumors, or degeneration of the submucous tissue, or infiltration of new-formed erectile tissue, sometimes occurs primarily.

Colloid cancer, affecting the stomach, behaves much as it does elsewhere; it originates, as the other species generally do, in the submucous tissue, and, as in a case excellently described by Dr. Walshe, may cause atrophy and destruction of the mucous membrane, over a more or less considerable space. We agree with this observer, that colloid may also be developed in the mucous membrane itself; for we have seen, in examining the mid-region of the stomach affected with scirrhus pylori, two large oval cysts, or cells, lined by a vesicular epithelium, and full of a clear fluid in the substance of the mucous tissue. It seems not improbable that these would have developed into a colloid growth, especially as the scirrhous formation contained loculi, somewhat similar to those described in the former observation. The mucous membrane, covering the cancerous growth, may undergo various changes. "It sometimes degenerates into an areolar cancerous tissue, which discharges large quantities of gelatinous mucous fluid; or it is converted into erectile tissue as a fungoid growth, which becomes the seat of encephaloid infiltration, suppurates, and partially exposes the submucous scirrhous cellular tissue; or, lastly, it most frequently becomes the seat of a sloe-black softening, with hemorrhage," or it is quite destroyed, and the sloughing process attacks even the denuded scirrhus itself. In a specimen we recently examined of scirrhus pylori, where the mass, limited to the pyloric region, was exposed on its inner surface, forming a sloughy ulceration with elevated, thickened margins, a fatty transformation had very evidently commenced. It was most apparent in the contents of the loculi, which, in some parts, consisted of well-formed nuclei and granulous matter, but, in many others, only of an amorpho-granulous substance, imbedding much oily matter. It is conceivable that the further progress of this change might have effected a cure. In this case it was very distinct; and it is worthy of remark, that, while the muscular coat had undergone very considerable hypertrophy, it was in no degree affected by the cancerous disease. This, though encircling the pyloric outlet, had not caused any actual obstruction to the passage, nor was the stomach distended in any very considerable degree. The

cause, therefore, of the hypertrophy of the muscular coat does not seem sufficiently explained.

A case is recorded by Andral, in which enormous dilatation of the stomach had taken place, although the pyloric orifice was free, and even larger than natural. He accounts for this by the non-existence of muscular fibres in this instance in the vicinity of the pylorus. Admitting this explanation, we are inclined to think that the very alteration of the natural condition of the outlet, its being reduced to a passive and rigid orifice, may necessitate a greater exertion of the muscular fibres, which, if it fails to take place, and thus induces a conservative hypertrophy, dilatation must result. Dr. Walshe, after mentioning the more usual occurrence of dilatation ensuing when the pyloric opening is obstructed, and contraction when the cardiac is, the size remaining unchanged when the body of the organ alone is affected, notices as "less intelligible" the fact to which we have just referred. He also remarks that, "as a general truth, the mucous membrane exhibits a notable power of resistance to the encroachment of the disease." This, we think, is true, at least as far as naked-eye investigation can ascertain; but in one case, where the mucous membrane appeared tolerably healthy, we found the tubular secreting structure in process of disorganization, not, however, from the extension of the cancerous disease.

It is necessary to be on one's guard against confounding scirrhous cancer with simple induration and hypertrophy of the coats of the stomach. Rokitansky enumerates as distinguishing signs the preponderating increase of substance in the submucous cellular tissue, and its want of uniformity, the accompanying cartilaginous hardness and closeness of texture, the fusion with the mucous and muscular coats, and particularly the alteration in the muscular tissue itself. We think the microscope, in practised hands, would generally clear up all doubt. When loculi of cell-substance are mingled with the fibrous tissue, there can be little hesitation in regarding the growth as cancerous. If the structure is purely fibrous, attention must be directed to the limitation of the disease, and to the existence of the infiltrating, softening, and contaminating properties of cancer. Ulceration, usually the result of secondary gastric cancer, may cause perforation of the stomach and fatal peritonitis; it more frequently happens, however, that effusion is prevented by the formation of adhesions between the threatened part and contiguous viscera. The liver and pancreas may thus become the seat of further cancerous invasion and destruction, or the ulcer may eat its way into the transverse colon, and thus cause an unnatural communication between its cavity and that of the stomach. A dark fluid, resembling coffee-grounds, is often found in the cancerous stomach after death, as well as vomited during life. In one case, where we examined it, we found it to consist of very numerous blood-globules, together with black granules and grains (probably altered hæmatin), and a very large quantity of amorphous, with some oily matter. It is to be remembered that vomited matter of this kind is not peculiar to cancerous disease; the same may be brought up when there is simple exhalation of blood from the mucous membrane, common ulceration, or follicular ulceration, or

even softening. The only circumstances necessary for its production are hemorrhage and the acid secretion of the stomach.

Besides blood, there may be several other matters abnormally present in the stomach. Unhealthy mucus in large quantities, purulent and other exudations, bile, biliary calculi, fecal matter, and lumbrici, are more or less often met with. Foreign bodies, of the most various kinds, are also to be included in the list, as the sealing-wax, brick-dust, cinders, &c. swallowed by hysterical females, or those who are subjects of the morbid state termed *pica*, or by actual lunatics. A remarkable case of this kind has been recorded by Mr. Pollock, in the Report of the Pathological Society for 1851–52, in which the stomach was distended by a large mass of hair and string, while another occupied the lower portion of the duodenum and commencement of the jejunum.

VI.—ABNORMAL CONDITIONS OF THE INTESTINAL CANAL.

The intestine is not unfrequently *defective* in some part of its course; this most commonly is the case near its lower termination, and involves an imperforate condition of the anus (*atresia ani*). Sometimes the intestine is only unusually short and of uniform caliber, or consists of several detached cœcal portions, or it may terminate at the umbilicus, or in a cloaca common to it and the genito-urinary organs. Andral refers to a case in which there was only a single straight canal, extending from the termination of the œsophagus to the commencement of the rectum, to another in which the duodenum was double, a third in which there were two colons, to a fourth in which the appendix vermiformis was unusually large, and at the same time double. All these, except the first, are instances of *excessive* development, though Rokitansky refuses to regard them as such, and considers them as "arrests of formation." Among these, he especially includes the *diverticula*, which are not very unfrequent, and which deserve a particular description. Andral compares them to the fingers of a glove, and states that they form cœcal appendages, one or more in number, which are given off from the intestine at various points, and communicate with its cavity. They are most frequent at from 18 to 24 inches from the termination of the ilium, according to Rokitansky, but have been seen in the jejunum, the duodenum, and even in the rectum. Their length is various; sometimes only a few lines, sometimes several inches; their cavity may be equal, greater or less than that of the intestine with which they communicate. It is most usual to find but one, but as many as six have been met with, originating from the same portion of intestine at a little distance from each other. In structure, they are sometimes identical with the intestine; sometimes their several coats appear to be hypertrophied; sometimes, on the contrary, more or less imperfectly formed. Meckel has founded on this difference a distinction of these diverticula into true and false. The false might be regarded as produced by a mere hernia of the mucous membrane, such as occurs in the bladder. True diverticula, Meckel considers to be formed by the non-closure of the vitelline duct at the usual spot, so that a portion of the canal, of varying lengths,

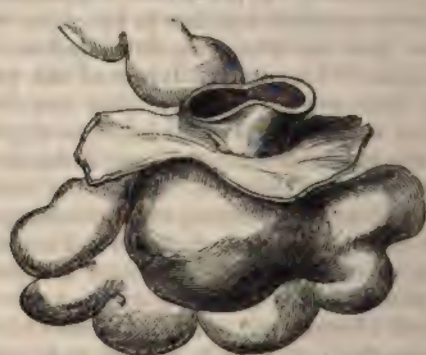
remains open after the intestine is fully developed. Rokitansky does not quite assent to this view, but still believes that "it evidently has its origin in the development of the intestine in the umbilical vesicle." We are inclined to join with Andral in doubting the correctness of Meckel's theory. It is not by any means shown that the multiple diverticula occasionally found are produced by a hernial protrusion of the mucous membrane. The distal extremity of these offsets is generally free, sometimes adherent to the parietes of the abdomen, or to the mesentery, or to a loop of intestine. Occasionally, it is open; Meckel met with an instance in which a diverticulum terminated at the umbilicus, leaving an orifice by which a probe could be introduced into the intestine. The omphalo-mesenteric vessels were persistent also, accompanying the diverticulum; so that in this case, at any rate, Meckel's view would appear justified. A case is recorded by Dr. Lionel Beale, in the Report of the Pathological Society for 1851–52, in which fatal peritonitis ensued from softening and perforation of the lower part of a diverticulum, which was twice as broad there as in the upper part adjoining the intestine. It is very conceivable that these offsets, like the appendix vermiformis, may become sources of danger by offering a favorable situation for the lodgement of cherry-stones, or other indigestible matters. Rokitansky gives the following description of the characters of "false diverticula," which he regards as mere herniæ of the mucous membrane, "resulting from the separation of the fibres of the muscular coat." They consist solely of mucous membrane and peritoneum. They occur at any part of the small and large intestines. "They are found in considerable numbers. They occur from the size of a pea to that of a walnut, in the shape of round baggy pouches of the mucous membrane. They form, more especially in the colon, nipple-shaped appendages, which occasionally are grouped together in bunches; when occurring in the small intestine, they are commonly developed on its concave side, and are therefore placed between the layers of the peritoneum; when in the colon, the feces are retained by them, and dry up into stony concretions." Uniform *dilatation* of the intestine may take place either from inaction of the muscular coat, or from distension from accumulation of its contents above a stricture. Disease of the nervous centres, inflammation of the serous investments, or simple atony of its contractile fibres, may be the cause of paralytic inaction and consequent distension. When a stricture exists, enormous dilatation sometimes takes place. Andral relates a case in which the large intestine was so distended that it resembled that of a horse, and concealed almost all the viscera. In such cases, the muscular coat may be in full and painful activity, not ceasing its action, although unable to overcome the obstruction, but reversing it so as to produce fecal vomiting.

Contraction of the intestine may occur either throughout a considerable extent, or in a very small one. The first is not a condition of disease, though it has been mistaken for such, and results merely from the canal at that part being empty for some time; so that the natural contracting efforts of the circular fibres are unopposed, and the sides of the tube are brought together. We have seen the descending colon in this way so shrunk as to be scarcely larger than the little finger. Of

course this condition is most likely to occur in the part of the intestine below a stricture. The second kind of contraction of the intestine is almost always morbid, and may result either from external pressure, as from a strangulating band, or compressing tumor, or from disease of the tissue in the part affected. The cicatrix of a simple or tuberculous ulcer, which had assumed an annular form, or a cancerous growth of like shape, has, in most instances, been the cause of stricture originating in the intestine itself.

Changes in the position of the intestine leading to morbid effects, require some particular notice. The most common of these constitute the several varieties of hernia. As these are fully described in all surgical works, it does not seem necessary to make mention of them here; and we shall, therefore, confine our attention, following Rokitansky, to the morbid changes of position which befall the intestine within the abdominal cavity. A portion of intestine may be twisted upon its own axis, so as to obstruct the canal by the approximation and contact of the walls. This seems to have been observed only in the ascending colon. The mesentery may be twisted upon itself, forming a kind of cone, with more or less of the intestine attached to its base, which becomes strangulated by being twisted round the mesentery. "One portion of the intestine, either single or double—a coil—may afford the axis round which another portion with its mesentery is thrown, so as to be throughout in contact with the circumference of the axis, and thus to compress it like a ferule. A coil of small intestine, the sigmoid flexure, or the cœcum, may form the axis." Abnormal length of the mesentery probably predisposes to these affections. More fre-

Fig. 217.



Strangulation of intestine by a portion of it slipping through an opening in the mesentery or omentum.

quent than the foregoing, are the instances in which a portion of intestine, generally the small, becomes strangulated by consequence of having got into one of the following situations: (1) into the fissure of Winslow; (2) into an opening in the mesentery; (3) into an opening in the omentum; (4) into spaces included by corded bands of false membrane, and various parts of the abdominal viscera; (5) into similar spaces formed by a long vermiform process, or intestinal diverticulum.

The colon and the rectum have been known to be compressed and obstructed by a mass of loaded small intestine lying upon them; and Andral refers to a curious case in which the transverse colon, in a child of six months old, was compressed between the duodenum and the vertebral column. These varieties of incarceration occur at every period of life; they are more common, Rokitansky avers, in the female sex than in the male, because the sexual organs of the former not only offer an additional point of attachment for constricting bands, but may also themselves give rise to constricting growths. The consequences of strangulation taking place in any of the above ways are distension of the intestine above the compressed part, peritonitis and ileus: the incarcerated portion in the cases where there is much pressure on the mesenteric vessels, is peculiarly liable to congestion and gangrene. Andral well remarks that the mere existence of the bands of adhesion, forming the orifices above described, by no means necessarily involves a strangulation of the intestine; this, however, in such cases, may come on very rapidly when the included portion of intestine becomes distended from any cause. In some cases symptoms are observed for several months or years, indicating that some impediment to the free passage of the intestinal contents exists. Dr. Peacock has related, in the Report of the Pathological Society for 1848–49, two cases of so-called mesocolic hernia; the small intestines being contained in a sac, formed by the layers of the mesocolon. In the first of these cases, there was no strangulation, though the ileum passed out of the sac over "a thin crescentic edge;" in the second, the ileum was strangulated at the part where it escaped.

Invagination of the intestine implies the inversion of a portion, and its intrusion into another, an upper portion being generally intruded into a lower, and the converse rarely occurring. The following account we abbreviate from the very full details given by Rokitansky. Invaginations not uncommonly form, during the last moments of life, especially in diseases which give rise to irregular and disturbed innervation. Thus, they are often met with in the dead bodies of children who have died from hydrocephalus. They are characterized by the absence of all appearances of reactions, such as we shall presently mention, and by the parts being easily restored to their proper situation. Several often occur in the same case. When they occur during life as a primary affection, or consecutive only to diarrhœa, they speedily bring the patient into a condition of extreme danger. Every intussusception must present at least three layers, as will be readily comprehended on viewing the accompanying diagram: the outer, called the sheath, is formed by a portion of intestine in its natural position; the middle is formed by the portion of intestine immediately above, which is inverted; so that its mucous surface looks outward, and is in contact with the mucous surface of the sheath; the inner is the portion of intestine next above, with its serous surface opposed to that of the middle layer, which is simply intruded into the canal beyond it. Five layers will exist if another portion of intestine be forced down, and inverted into the original intussusception, which then becomes a sheath to it. "Between the middle and inner layers there is a portion of mesentery

corresponding in size to that of the intestine displaced, and folded up so as to represent a cone, the apex of which lies at the free termination of the volvulus, with its base in the sheath, and at the entrance to the invagination. This portion of mesentery is always in a state of tension, which chiefly affects the part belonging to the inverted tube, and has a singular influence upon the form of the volvulus. It is the cause of the following circumstances: Firstly, that the volvulus (the middle and inner layers) does not lie parallel to its sheath, but always offers a greater curvature than the latter; the inverted tube (the middle layer) being compressed in its concavity into tense transverse folds. Secondly, that the orifice of the volvulus does not lie in the axis or in the centre of the sheath, but external to it; and that, following the traction exerted upon it by the mesenteric fold that belongs to the inverted intestine, it is directed towards the mesenteric wall of the sheath; that it is not circular, but represents a fissure. Intussusceptions occur with equal frequency in the colon, and small intestine; but several cases occurring in the former are remarkable on account of the magnitude they attain. In these, the sheath contained a very long portion of the colon and ileum; both may be inverted two or three times, and the intussuscepted part advances to the vicinity of the anus." Contraction of one part and distension of another, are probably the conditions which give rise to invagination; the upper contracted portion, whose muscular coat is for some distance upwards in a state of activity, being propelled onward into the dilated part below. Disordered and irregular innervation is probably the remote cause, in this as well as in the case of intussusceptions taking place during the agony. Increase of the invagination always takes place by more and more of the intestine becoming inverted, so that the upper border of the middle layer is continually shifted lower down. The consequence of invagination is, as may be expected, peritonitis and its results. Inflammatory congestion is set up, not at first, in consequence of annular constriction of the volvulus, but from compression of the vessels; especially the veins of the portion of mesentery which is dragged down by the advancing and inverted layers. This obstruction to the circulation, Rokitansky says, gives rise to violent inflammation with "plastic effusion on the contiguous serous surfaces of the entering and receding (inverted) tube, and upon the mucous membrane of the latter. The inverted portion is invariably the one that suffers most;" "and it is characteristic, that, even when the inflammation of the volvulus runs high, its mucous membrane remains pale." The sheath is not so much affected except in large invaginations. The tumefaction resulting from the inflammatory congestion may cause strangulation of the volvulus, usually at the entrance, but sometimes at other points. If death does not result from the peritonitis, the ensuing gangrene, or the strangulation, recovery

Fig. 218.

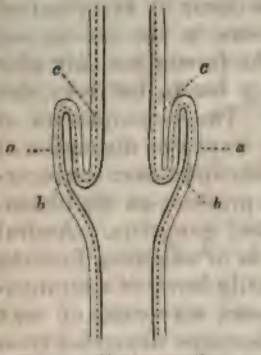


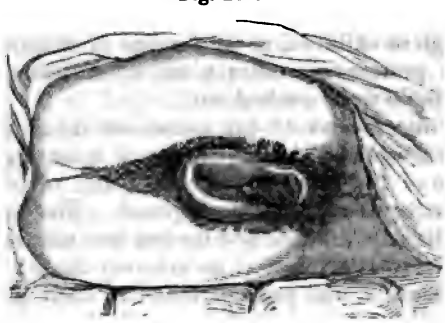
Diagram of intussusception.

(a) The sheath.
(b) The receding or inverted tube.
(c) The entering tube.

takes place with one of the three following terminations to the morbid process: (*a*) Complete adhesion having been formed between the two opposed serous surfaces of the sheath, and the inverted tube at the point of inversion, the whole contained volvulus mortifies and is thrown off, and becomes discharged *per anum*. In a case referred to by Andral, the portion of intestinal tube thus evacuated, measured eighteen inches; and Hévin relates two cases, in one of which twenty-three inches of the colon were thus parted with, and in the other twenty-eight inches of small intestine. An annular swelling, more or less interfering with the canal of the intestine, is found in the corpses of persons in whom this termination has occurred, besides adhesions of the serous surfaces in the vicinity. (*b*) "In rare cases, in which the incarceration has been developed at an unusual point, only a partial sloughing of the volvulus takes place; and the portion which lies above the strangulation is retained. Under these circumstances, the latter forms a conical plug with a narrow channel, and projects into the cavity of its sheath, surrounded by a thick fringe of mucous membrane." (*c*) Occasionally, after adhesion has taken place, the inflammation abates, and the volvulus is retained. The first mode of termination is the only one that produces a permanent recovery; in the others, there always remains a degree of chronic hyperæmia, with liability to exacerbation. No age is exempt from the occurrence of invaginations. Andral quotes from Monro a case of a very considerable one, in a child four months old.

Prolapsus ani is an affection very analogous to invagination, differing chiefly in not having a sheath, as Rokitansky remarks, and also, as we think, in the peritoneum being less involved. In trifling cases, only a

Fig. 219.



Prolapsus Ani.

fold of the mucous membrane comes down, but in the more serious, both the mucous and muscular tunics descend. The tumor thus formed is of a sausage or pyriform shape, pediculated by the contraction it undergoes at the anus, and having at its extremity, in the minor degrees, a round central orifice, and in the greater, an eccentric fissure. The results of prolapsus ani (more properly *recti*), are not, for the most part, nearly so serious as those of invagination. In some cases, indeed, strangulation takes place, the everted part swells to twice or thrice its proper

size, assumes a red, purplish color, with an appearance of ecchymosis and of impending gangrene. More often, even in inveterate cases, there is only a discharge from the irritated or mucous membrane of a thin muco-sanguineous fluid, with, perhaps, some superficial ulceration. In some cases the epithelium of the mucous surface, from constant exposure, assumes a cuticular character. The affection is of common occurrence in children, for which Sir B. Brodie assigns the following reason: "The attachment of the rectum to the surrounding part does not extend so high in children, as in persons of mature age; while the reflection of the peritoneum takes place lower down; and hence the rectum is more liable to be pushed out."

Fig. 220.

Prolapsus Ani.

Wounds and *lacerations of the intestines* demand a brief notice. When the bowel is wounded by the thrust of a cutting instrument, the danger of escape of the intestinal contents, and of consequent peritonitis, is not so extreme as might be supposed. The mucous membrane being somewhat loosely attached and thrown into natural folds, protrudes at the orifice so as to close it, if it be not very large; at the same time the constant pressure of the parietes tends to keep the wounded point applied against the opposing surface, to which, moreover, it becomes glued before long by adhesive exudation. If an opening is made into the intestine by ulcerative perforation of its wall, the effusion of lymph uniting it to adjacent parts becomes occasionally the medium through which a fistulous communication is established, either with the exterior of the body, or with another hollow viscus in the vicinity. Sometimes the ulceration, instead of penetrating further, causes simply the formation of abscess in the sub-peritoneal cellular tissue, fecal abscess, as it is termed. When the intestine is divided completely across, the two ends will occasionally unite, if brought together and maintained in apposition by stitches; but no one has yet succeeded in repeating Ramdohr's experi-

ment (said to have been successful), in which a large portion of an intestine having mortified, was cut off, and the upper end inserted within the lower, and kept in that position by a suture. It is very unlikely, that a mucous and a serous membrane would unite by adhesions, which is said to have occurred in that case.

The muscular coat of the intestines is very rarely, if ever, primarily the seat of inflammation, though it very often is involved by extension of the mischief from the serous or mucous tunics. We have, therefore, now to consider chiefly the condition of the mucous membrane and its follicles when inflamed. It must be premised that no kind or amount of vascular injection can be accepted by itself as a decisive proof of the existence of inflammation. Obstruction to the free return of blood by the veins, according to the degree in which it exists, will produce ramiform, patchy, or general injection; and the same cause will also give rise to the punctiform, which has been thought more characteristic of active hyperæmia. Very marked injection also results from mere gravitation of the blood after death to the most depending parts. MM. Trousseau and Rigot found in dogs, which had been suspended after strangling in a vertical position, the blood collected in those parts of the intestines which were the lowest, giving the mucous membrane and its villi a deep red tinge, and extravasating itself on the surface and in the submucous tissue. When the bodies were suspended in the reverse position, the same effects took place in those that were then the lowest. This hypostatic congestion is especially liable to take place in fevers, in severe cases of which, the blood is so commonly found fluid after death. Redness, resulting from mere staining of the tissues, is sometimes very similar to that of active hyperæmia. The injection which takes place from engorgement of the veins may be distinguished from that of inflammation, by the circumstance of its being always traceable to distended veins, and, further, we think, by the redness being more general than that of inflammation commonly is. In general, the smaller and more isolated the patch of injection is, the more likely is it to be inflammatory; thus, we find small separate patches of injection around commencing typhous deposits, which contrast with the generally pale mucous membrane.

Inflammation, affecting chiefly the mucous lining of the intestines, was distinguished by Cullen as *enteritis erythematica*. It corresponds to the muco-enteritis of others, especially the French pathologists. Rokitansky speaks of it as catarrhal inflammation, which may be either acute or chronic, and either attack the mucous membrane uniformly or be developed mainly in the villi and the follicles. It is excited by various causes of irritation, and especially by certain atmospheric influences. "The anatomical signs of the *acute form* are, more or less intense redness and injection of the mucous membrane, affecting its entire surface, or appearing as punctiform reddening from affection of the villi, or as a vascular halo surrounding the follicles; relaxation of the tissue, and intumescence of the mucous membrane, equally affecting the entire substance, or only the villi and follicles; opacity of the mucous membrane and its epithelium, from infiltration of the former and softening of the latter; friability and softening of the mucous membrane. The sub-

mucous cellular tissue is injected, relaxed, and infiltrated with a watery, opaque fluid; the secretion is at first copious and serous; as the affection increases in intensity, the former diminishes in amount, becomes opaque, viscid, and puriform.

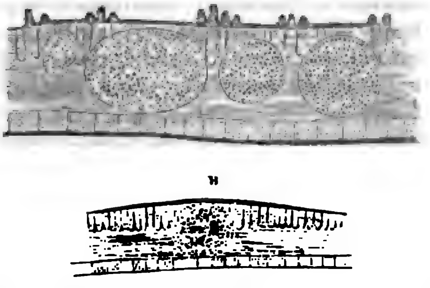
Chronic inflammation is characterized, in addition to the above signs, by a dark, rusty, livid discoloration, which, in severe cases, appears to pervade the entire mucous membrane; by a tumid state of the mucous membrane and its follicles, accompanied by increased density of the tissue, copious secretion of an opaque grayish-white or yellow puriform mucus. The acute form may subside completely, or several relapses occurring, it may merge into the chronic, which seldom "admits of a complete cure." The following are the principal changes produced by chronic intestinal catarrh: (1.) A more or less abundant deposit of black pigment in the whole mucous membrane, or in its villi or follicles only. (2.) A permanent tumefaction of the mucous membrane, depending probably on dilatation of its vessels and interstitial exudation, which causes increased density of its tissue, and may give rise, "in higher degrees, to elongation of the membrane, and formation of folds and polypi." (3.) Hypertrophy of the submucous cellular tissue and of the muscular coat. (4.) Profuse secretion of a grayish-white and milky, or of a transparent gelatinous and viscid mucus. Suppuration and ulceration sometimes result from catarrhal inflammation, especially when an acute attack supervenes upon the chronic form. "The mucous membrane is converted into a dark-red, granulated, and friable tissue, on the surface of, and within which, suppuration is established." The burrowing of the ulcers, so as to pass through even the muscular coat, gives rise to sinuses, in the vicinity of which there is often a production of polypoid growths from the mucous membrane. Corrugation of, and pigmentary deposit in the tissues, are constant accompaniments of this process. Cicatrization of the ulcers and sinuses takes place, with formation of the usual fibroid tissue, which, by its subsequent contraction, may give rise to puckering or obstruction. Rokitansky states that the most usual seat of catarrhal inflammation is in the cœcum and rectum; we doubt if this is true, if applied both to the acute and chronic forms, for there can be no doubt that, in the majority of instances of muco-enteritis, the small intestines are affected even to a greater degree than the large. He observes himself that "catarrhal irritation, and even inflammation, undoubtedly often affect the duodenal mucous membrane, and are frequently induced by an anomalous condition of the bile." This condition, as Dr. Stokes has observed, "may extend to the biliary ducts and give rise to jaundice, an instance of which we have lately had under our care. Besides the tumefaction of the mucous membrane, the increased pouring out of mucus, and the enlargement of the glands of Brunner in this situation, we have noticed an atrophy or destruction of the villi, which had lost their sharp exterior margin of limitary membrane, and were shreddy and wasted. The chronic form of catarrhal inflammation does seem to be more frequent in the large intestine, at least the slaty and black discoloration is more often seen in the cœcum than elsewhere; but it may admit of some doubt whether this discoloration is a certain sign of previous inflammation, whether a deposit of pigment may not take place here as in the

areolar tissue of the lung, or in the skin, without any disease. We have certainly examined instances in which the microscope showed nothing the least abnormal except the pigmentary deposit.

It seems very doubtful whether there is a distinct affection such as that which has been termed glandular or follicular enteritis. Dr. Copland speaks of it as occurring almost always consecutively to other diseases, as fevers, continued or remittent, dysentery, and even tuberculosis. Rokitansky does not seem to recognize its special character, but to consider that the follicles may be more particularly affected in morbid processes of different kinds. In this opinion we entirely coincide, but wish to notice here an anatomical point which we think is not generally understood. The solitary glands of the intestine, which occur both in small and large, as well as their aggregations, constituting the patches of Peyer, which do not extend beyond the ilio-cœcal valve, are quite destitute of the follicular character, that is, are not involutions of the general mucous surface, invested by a lining of epithelium. They are simply solid aggregated masses of nuclear particles, with very little

Fig. 221.

A



B

Vertical section of Peyerian patch, and solitary gland of large intestine. The glands in both are rather enlarged. (A) Peyerian patch from ileum. (B) Solitary gland.

intervening granulous matter, which lie completely beneath the sheet of basement-membrane that covers the surface. They are not contained in a distinct capsule of homogeneous membrane, but lie partly in the corium of the mucous tissue, partly in that layer of nuclei and granulous matter which is spread under the basement-membrane, and forms the chief substance of the villi, to which we gave, some years ago, the name of "substratum." It is apparent, from this structural arrangement, that they cannot be secreting organs like the Lieberkühn tubes all round them, for they have no outlet. On the other hand, they are, from their very structure, peculiarly liable to become enlarged and prominent, the nuclei attracting to themselves plasma, and developing into cells. There seems no doubt that they are much more developed in some persons than in others, and we are much inclined to think that masses of precisely similar appearance may be formed in the mucous membrane, solely as the result of irritation. We have been led to this belief from having found only a few distant and widely scattered glands in the mucous

membrane of some persons, even when it was examined after immersion in acetic acid, which makes them much more opaque and white, while in other cases they were extremely numerous and close together. There really seems to be a good deal of resemblance between them and small pustules of the skin, like which, when the inflammation reaches a certain point, they are extremely apt to suppurate and slough. We shall now subjoin Rokitansky's account of "ulcerative inflammation of the follicles of the colon, such as we find in lientery, brought on by tedious diarrhœas. The follicles are at first tumefied in various degrees, and consequently project as smaller or larger round, conical nodules on the internal surface of the intestine, being surrounded by a dark-red vascular halo. Ulceration now ensues in the interior of the follicle, the small abscess penetrates the mucous membrane within the vascular halo, and a fringed ulcerated opening, of the size of a millet-seed, appears, which leads to a small follicular abscess with red spongy walls. The ulceration continues, and the follicle is eaten away," so that an ulcer of the size of a pea or lentil is formed. In its further progress, the mucous membrane becomes extensively destroyed, and that with great rapidity, the muscular coat being frequently exposed. The most extensive destruction always takes place in the sigmoid flexure and in the rectum. The disease is always confined to the colon; but, when it runs a very rapid course, it may be accompanied by catarrhal irritation of the small intestine. We have notes of a case in which there were a great number of the so-called solitary follicles in the lower part of the small and in the large intestine, although no irritation had existed during life. Their development seems to depend on some other cause besides mere irritation, though this, no doubt, promotes it. Like the Peyerian patches, they are sometimes very prominent in cases of typhus, sometimes scarcely discernible; and the same is true of common catarrhal irritation and of dysenteric. It can scarcely be supposed that the Lieberkühn follicles, which are mere involuted extensions of the mucous surface, should not be affected wherever the mucous membrane is; but the solitary glands, which differ essentially in structure, seem to be by no means necessarily involved.

We proceed to the other form of enteritis, that termed by Cullen, E. Phlegmonodea. The effects produced by this, which, indeed, are just those of common inflammation, have already been several times adverted to; but we think it well to subjoin the following excellent description from the pen of Dr. Copland: "The villous coat in acute enteritis is not only more vascular and turgid, but it is also softer, and sometimes thicker than natural. If the inflammation has proceeded far, it presents a brick-red tinge, and is easily detached from the subjacent coats, the connecting cellular tissue being soft, turgid, and inflamed. When this state exists in a considerable portion of the tube, the coats are apparently thickened, arising from the extension of the inflammation to the more external tissues, till the attached surface of the intestinal peritoneum is reached. The substance or parietes of the bowels may be considered as affected in these cases, even although the external surface may present no further lesion than red vessels shooting into it. Occasionally, in addition to this state, the red capillaries in the inflamed

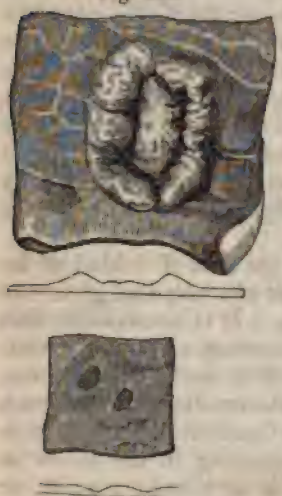
peritoneal coat are connected with the effusion of coagulable lymph, particularly in the parts where they are most numerous, the lymph or albuminous exudation existing in specks, or in considerable spots or patches, on the serous surface. When, however, these latter appearances are remarked, the interior of the inflamed intestine frequently presents more serious changes than yet noticed. The villous surface is then deeply inflamed, and seems abraded or excoriated in parts. It is sometimes, in other parts, covered by patches of lymph, or of an albumino-puriform, or muco-puriform fluid, or by a sero-sanious matter; and it is often also ecchymosed in numerous points or specks, or it presents still larger marks of sanguineous infiltration. In other cases, portions of a dark, slate-colored, or sphacelated hue are observed, with or without ulcerated specks, or even large ulcers, which have nearly penetrated as far as the external coat, in adjoining parts." Sometimes then ulcers actually perforate the intestinal wall, and give rise to escape of the contents. "In the forms of enteritis in which the substance of the intestine, or its peritoneal coat, is chiefly affected, either primarily or consecutively, the whole of the coats are often very vascular, red, or of a brick-red color, and are readily torn." Suppuration sometimes takes place between the coats, as in an interesting case recorded in the Pathological Society's Reports, 1851-52, by Dr. Hare. General peritonitis not unfrequently occurs, in which the omentum may markedly participate, becoming greatly thickened and red.

The intestinal mucous surface is sometimes the seat of a kind of inflammatory process, which is mostly subacute or chronic, and gives rise to an exudation much resembling that of croup. It is remarkable that the attacks often recur several times, each presenting a stage of irritation, which ends in the formation and throwing off of the false membrane, after which there is a pause. The quantity of the exudation varies much in different cases; sometimes it forms a layer of some thickness, extending pretty uniformly over the surface, or appearing in the stools as tubular casts of the intestines; sometimes it is as thin as a wafer, or consists merely of tattered shreds. In one case, mentioned by Dr. Copland, there were also shreds of dysmenorrhœal false membrane discharged from the uterus, but not at the same time as those from the bowels. Rokitansky mentions the occurrence of less consistent fibrinous exudations, which probably approach more or less closely to those of diphtheritis. They are, as he observes, "the expression of a constitutional affection," the results of an altered blood crasis.

The changes which take place in the intestinal mucous membrane in continued fevers have been most diligently examined, and minutely described by Rokitansky; but our limits forbid us to follow him closely, and we shall, therefore, only endeavor to give a short and comprehensive account of the various phenomena belonging to what he calls the typhous process. The intestinal affection, as is well known, is no necessary part of fever. We have examined the intestines of persons dying of fever, in which very little trace of Peyerian patches or solitary glands could be detected, certainly less than we have seen in the bodies of persons who have died from other diseases. When, however, the malady specially affects this seat, we observe the following series of changes: Hy-

peræmia, to a greater or less extent, is set up around the solitary follicles, and in and around Peyer's patches. Enlargement and distension of these glandular structures proceed nearly *pari passu* with the hyperæmic congestion. After a certain time, the length of which varies in different cases, the contents of the enlarged glandular masses soften, break down, and are discharged. The cavity which remains on the mucous surface, constitutes the typhous ulcer, to which Rokitansky attributes the following character: (1.) Its form is elliptical, round, or irregular, and sinuous, according to the shape of the part which has been affected. Thus, a large patch, when destroyed, gives rise to an elliptical ulcer; a smaller or a solitary gland to a round one; partial destruction of a patch will produce an irregular ulcer. (2.) The size of the ulcer varies from that of a hemp-seed to that of a half-crown. (3.) Those of an elliptic shape are always situated opposite to the insertion of the mesentery, and have their long axis parallel to that of the intestine. The typhous ulcer very rarely indeed forms a zone. (4.) "The margin of the ulcer is invariably formed by a well-defined fringe of mucous membrane, which is a line or more wide, detached, freely movable, of a bluish-red, and subsequently of a slaty or blackish-blue color. (5.) The base of the ulcer is formed by a delicate layer of submucous tissue which covers the muscular coat: like the marginal substance, it is quite void of morbid growth. (6.) The small intestine is the seat of the ulcerative process, and the lower third is most liable to be involved—the number and size of the ulcers increase as they advance towards the ilio-cœcal valve." We must add with respect to the last character, that the ulcerative process is by no means confined to the small intestine; we have seen the mucous membrane of the large intestine, down to the rectum, riddled with ulcers. They were many of them of large size, and had clean cut, non-thickened margins. This condition, indicating the absence of reparative action, is not nearly so frequent as that of thickening and induration, which generally takes place to some extent in the side of the ulcer. The bottom of the ulcer is commonly formed by the submucous tissue, sometimes the muscular fibres are completely exposed. This, however, is generally the result of secondary advance, subsequent to the expulsion of the typhous deposit. Rokitansky particularly insists on the point, and we think he is correct, that when an ulcer increases in depth so as to perforate the intestine, it advances not by continued deposition and softening of typhous matter, but by simple extension of the ulcerating action. We feel inclined to doubt Rokitansky's statement respecting the extreme rarity of a typhous ulcer assuming a zonular form. We think we have seen some that in-

Fig. 222.



Typhous ulcers in small intestine. Death from hemorrhage. The outline figures represent vertical sections. In the upper figure the margins of the ulcer are thickened; in the lower they are clean cut, as if punched.

volved a considerable part of the circumference of the intestine, and Andral mentions a case in which there were a dozen ulcers encircling the canal like rings. It is true, he does not positively state that they

Fig. 223.

Inflamed mesenteric glands in Typhus and so-called typhous matter. At the lower and left part is represented a small ulcer in the mucous membrane, in which ulceration is seen extending round the central typhous deposit, which is still *in situ*.

were the result of fever, though this seems almost implied. The matter which causes the tumefaction of the agminate and solitary glands is

Fig. 224.

Typhous ulcers in various stages. The outline figures are vertical sections, which show the elevation of the mucous membrane by submucous deposit. (*a.*) Mucous membrane. (*b.*) Submucous tissue. (*c.*) Muscular coat. (*d.*) Peritoneal coat.

simply a kind of albuminous exudation, not differing we believe essentially from any other. It either, which is most frequent, forms a solid mass imbedding the natural nuclei of the gland, or affords a plasma, out

of which these nuclei develop celloid particles. Black granules and grains of pigmentary matter are often present in it, but they are by no means peculiar to the typhoid state. They give to the glands a black dotted appearance, as seen by the naked eye, and this we have observed more than once after death from other causes than fever. The mesenteric glands become invariably enlarged in all cases of intestinal affection, just in the same way as an inguinal gland enlarges when there is a chancre on the glans penis. Their enlargement seems to be simply the result of irritation ; we have found nothing in their substance besides the normal nuclei but granular and amorphous matter, and some celloid particles or cells. The vessels of their capsule are generally much congested, as well as those which penetrate their interior. Rokitansky states that "the mesenteric glands decrease in size, as soon as the detachment of the intestinal morbid growth has commenced." Of course, they must remain for some time more congested with blood, and larger than natural, even under the most favorable circumstances. It by no means necessarily follows that ulceration takes place after a patch or a single gland has been enlarged; the exudation may liquefy and be again absorbed into the blood, and the part return to its normal condition. Cicatrization of the ulcers is not unfrequent, as Dr. Watson testifies. He says: "The ulcerated surface seems to clothe itself afresh, by degrees, with a new mucous membrane, which is thin, however, and adherent to the subjacent tissues, and does not slide over them when pressed between the finger and thumb, as the healthy portions of the coats of the bowel will do upon each other. And in the place of the cicatrix there is usually to be seen a manifest puckering, and a number of little wrinkles or lines, radiating from a common centre."

Rokitansky speaks of the new-formed membrane as a serous lamina, which becomes at its circumference as it were dove-tailed in between the muscular and mucous coats. He confirms the observation of Sebastian, that small villi sometimes form upon this lamina, even before its union with the mucous membrane. In most instances, however, the absence of villi forms one of the distinctive features of a cicatrix. We feel much hesitation in accepting Rokitansky's absolute assertion, that the cicatrix of a typhus ulcer never in any way gives rise to a diminution of the caliber of the intestine. Dr. Carswell speaks positively of the occurrence of fatal ileus in persons who had suffered some months before from typhoid fever, the cicatrix of an ulcer being found after death, which had destroyed the muscular coat around the whole circumference of the tube.

The morbid changes in *Dysentery* have their especial seat in the large intestine, the ileum being sometimes (Dr. Copland says very often) involved, but always in a less degree. Our observation quite accords with Rokitansky's, that "as a rule its intensity increases from the cœcal valve downwards," so that the sigmoid flexure and the rectum are found most severely affected. It commonly runs, Rokitansky says, an acute course, "though it is frequently chronic in the milder degrees; this, however, does not materially alter its character." Dysentery presents itself to the medical observer under a very great variety of forms, but it would be impossible to range the post-mortem appearances

in corresponding groups. All that can be said in general, is that both the symptoms during life, and the textural lesions, will coincide in indicating whether a given case is to be considered of sthenic or asthenic character. Looking, then, upon dysentery generally as inflammation, more or less acute, of the mucous membrane of the large intestine, and premising that it is very prone to pass on into a lingering chronic state, we shall follow Rokitansky's account of the changes produced by the disease. He considers them "as divisible into four natural degrees." In the lowest, the mucous membrane in its projecting folds is injected, swollen, and softened, its surface seems excoriated, the epithelium is detached as a grayish-white layer, or elevated by effusion into small vesicles containing serum, as in a case we witnessed, or it may be mingled with amorphous matter in an exudation of dirty gray and reddish color covering the surface. Dr. Baly, who mentions the detachment of the epithelium and its mingling with amorphous matter, describes the solitary glands in his first stage as being enlarged, forming round prominences, which, in a chronic state, by sloughing and ulceration, assume the form of open sacs. The mucous and submucous coats become thickened. "In the second degree, the textural alterations are not limited" to the projecting folds, "but extend over a larger surface, still, however, presenting a greater development at one part than at another." The mucous membrane is invested to the same extent, by a dirty, gray layer, consisting of desquamated epithelium and a thick glutinous exudation; or this may already have been removed, and the subjacent mucous membrane, in either case, appears converted into a soft, sanguineous, pale red and yellowish gelatinous substance, which may be easily detached." The submucous tissue becoming infiltrated, gives rise to more or less numerous protuberances, on the internal surface of the intestines; these "correspond to those points of the mucous membrane, at which the morbid affection is most developed," while in the intervening portion, there is not much change beyond slight redness and swelling. The intestinal cavity is dilated by the pressure of exhaled gas upon the semiparalyzed muscular coat, and contains a mixture of effused lymph and blood, together with mucous liquid and feces. Dr. Baly marks his second degree by the sloughing of the solitary glands, either principally or equally with the surrounding mucous membrane. In this way are formed either clear circular ulcers of various depth, or large excavations. The prominent rugæ are chiefly affected. Rokitansky also remarks, that the affection of the follices may predominate, and states that the anatomical condition is the same as that already described in connection with catarrhal inflammation as attendant upon lientery. "In the third stage, the protuberances are set more closely together, the mucous membrane investing them partly retains the same condition as in the former, partly" is converted into a slough, which is here and there blended with the desquamated epithelium "and the exudation, and is firmly attached to them; it is of a dark-red, or blackish-brown, or grayish-green color." In some cases, the infiltrated and thickened submucous tissue is in great part exposed, being covered here and there by the remnants of the mucous membrane, "in the shape of solitary, dark-red, flaccid, and bleeding vascular tufts, or as dilated follicles, which

are easily removed." The protuberances are occasionally found to have coalesced, and the intestine then presents an uneven plicated surface, accompanied by an equal degree of infiltration and thickening of its parietes; the mucous membrane is uniformly affected over a large extent, and there are no free interstices. The contents of the intestine are of a dirty brown or reddish, ichorous, fetid, flocculent, and grumous character.

In the fourth and highest degree, the mucous membrane degenerates into a black, friable, carbonified mass, which may often be subsequently voided in the shape of tubular laminæ (so-called mortification of the mucous membrane). The submucous cellular tissue appears to be previously infiltrated with carbonified blood, or a sero-sanguinolent fluid; or it is pallid, and the blood contained in its vessels is converted into a black, solid, or pulverulent mass; subsequently, it shows purulent infiltration, in consequence of the reactive inflammation which is induced in the lower, healthy strata, for the purpose of eliminating the gangrenous portions. Dr. Baly's third degree seems to correspond to the fourth of Rokitansky; he describes the mucous membrane as converted into a gangrenous slough, glands and all alike, the blood being dark and coagulated in the submucous tissue. The prominent rugæ are first and most severely affected, the intervening portions being swollen and red. All the coats sometimes are much softened, and the submucous tissue becomes sloughy. The changes just enumerated as occurring in the most extreme degree of the disease, are of much the same kind as those which Dr. Copland describes to take place "in the most malignant varieties, and in the scorbutic complication. The internal surface of the whole digestive tube is," in these cases, "of a livid purple, or dark color, with patches of ecchymoses, excoriation, ulceration, and sphacelation. The villous coat, particularly in the seat of ecchymoses, may readily be rubbed off; and the ulcers have a foul and dark appearance. The liver is sometimes large, soft, and spongy; at others, pale and soft, especially in cases where the loss of blood has been very large. The spleen is sometimes so softened as to appear semi-fluid or sphacelated. The heart is often partially softened or flaccid; the pericardium and pleural cavities containing a bloody, dark, and dirty serum. The lungs are often congested; the bronchial lining dark or ecchymosed; and the blood, in all the large vessels, is semi-fluid, black, and of a very loose texture." "In prolonged inflammatory cases, thickening and almost cartilaginous induration of a considerable part of the colon are not unfrequent, the thickened or indurated portion being also contracted in caliber. In such cases, the parts above the contraction are greatly distended, the coats being thinned, ulcerated, and even lacerated;" so as to give rise to effusion and fatal peritonitis. The darkly-congested mucous membrane is often discernible through the peritoneal and other coats, especially if, as is often the case, the intestine is distended. In the severer cases, the serous membrane is dulled and discolored, and sometimes covered with a brownish ichorous exudation. The mesocolic lymphatic glands are swollen and congested. In the dysentery which occurs in this country, abscess of the liver is rare; a case, however, is recorded in the Report of the Pathological Society, 1851–52. Dr. Baly

has never met with it during his experience at the Millbank Penitentiary, where the disease is very common; but in India, it is said to occur in nearly half the cases. We have recorded, in the Report of the Pathological Society, 1847–48, an instance in which the destruction of the mucous membrane was confined to the interstices of numerous prominences or ridges, these being themselves the sole remnants of the mucous tissue. This is the converse of the more usual condition, in which the prominent parts are most affected. After extensive ulceration has occurred, reparation may be effected in the usual way by organization of plastic exudation into a smooth fibroid layer which constitutes a cicatrix. This probably may be covered after a time by an epithelial layer; but it has not been shown yet that the follicles of Lieberkühn are reproduced. When extensive destruction of substance has taken place, the cicatrix tissue "is frequently condensed into fibrous bands, which form corded projections into the intestinal cavity, interlace with one another, and not unfrequently encroach upon the caliber of the intestine in the shape of valvular or annular folds, thus giving rise to a stricture in the colon of a very peculiar form." Instead of reparation taking place, the disease may continue in a chronic though altered form; the remaining mucous membrane being in a state of chronic catarrhal inflammation, and the intervening parts being the seat of suppuration with formation of sinuses and abscesses.

Gelatiniform softening, analogous to that described as affecting the stomach, occurs also in the intestines, but much less frequently. "It involves," Rokitansky says, "the external coats, converts them into an homogeneous, grayish-red, transparent, and deliquescent gelatin, and leads to spontaneous perforation." It is far more often met with in children than adults, and seems to be occasioned by unsuitable food, bringing up by hand, and generally by causes which depress the organic nervous power; diarrhœa, absence of fever, and great and increasing debility, are the prominent symptoms. Dr. Droste, quoted by Dr. Copland, distinguishes three stages of the softening process; in the first, the villous surface retains its texture, but loses its consistence more or less extensively; in the second, it is converted into a thin, soft, gelatinous and nearly transparent substance, capable of being washed away; in the third, "no trace of organization is left in any of the coats," which are perforated, or on the point of being so, in various places. *Fatty tumors*, sessile or pediculated, occur in the intestinal canal; they originate in the submucous tissue, where fat-vesicles always exist, and grow inwards. *Serous* and *fibro-serous cysts* are met with but very rarely between the intestinal coats. *Fibroid nodules*, not exceeding the magnitude of a pea, are sometimes found in the submucous tissue. *Calcareous concretions*, formed by the deposition of earthy matter in new-formed fibroid tissue, in obsolete tubercle, or desiccated pus, or fibrinous exudation, occur very rarely. Rokitansky describes *erectile growths* to exist in the intestinal canal, either in the form of sessile tumors, or pediculated polypi. We are not sure whether he would include under this head instances of fibrous polypi, such as two recorded by Mr. P. Hewett, in the Report of the Pathological Society for 1846–47, which, though of marked fibrous structure, were livid in appearance, and pretty plenti-

fully supplied with bloodvessels. The presence of these polypi may give rise to invagination and its consequences. *Tubercle* selects the small and large intestines not unfrequently as its seat of deposit. It is met with more frequently in children than in adults, in the proportion of sixty-one to forty-three, and is more than twice as frequent in the small as in the large intestines. In the majority of cases the affection of the intestines is secondary to that of the lungs, and usually takes place after the tubercles in the latter have begun to suppurate, and the cachexia has become fully developed. The course of intestinal tuberculosis is "frequently chronic, but much oftener acute." The seat of the deposit is the submucous tissue, or the corium of the mucous membrane; it is certainly subjacent to the basement-membrane, and not contained in the follicles, as Dr. Carswell taught. Rokitansky states that there is, in the chronic form, "no perceptible inflammatory action, and the disease appears in the shape of the gray transparent granulation, which softens at its centre, and is gradually converted from within outwards, into the yellow cheesy tubercle. It seems blended with the mucous membrane, and projects into the intestinal cavity in the shape of a sessile, hard nodule. Considerable inflammatory action attends upon acute tuberculosis. The exudation affects first Peyer's patches, then the solitary glands, and, lastly, every other part of the mucous tissue; it appears in large masses, and in the shape of yellow cheesy matter, which speedily undergoes a purulent transformation. The surrounding tissue is extensively congested, reddened, and turgid, offering a good illustration of the influence of hyperæmia in promoting tuberculous exudation. In the great majority of cases, as the tubercle softens, the mucous membrane over it gives way, and the suppurating mass escapes. In very rare cases this does not take place, but there is formed a small abscess in the submucous cellular tissue. The margin of the tubercular ulcer is firmly attached, rounded, and indurated; its base, usually formed by condensed areolar tissue, may contain or not tuberculous matter. The enlargement of the ulcer in depth and in extent is effected by a process of essentially the same kind as is observed in cavities of the lung. The margin and the base become infiltrated with tuberculous exudation which softens and suppurates, and so the destructive process continually advances. Perforation of the intestinal wall sometimes occurs, the ulcer retaining throughout its original character, in which respect it differs from the typhous. The fatal event is often prevented by timely exudation of fibrin on the serous surface, which either thickens the wall or else unites it to an opposing surface. Mr. Ancell contests the statement that the tuberculous ulcer always retains its original character; he does not believe that ulcerations, the result of tuberculosis, are always produced by the deposit of tubercle. He views them as the result of "the ulcerous diathesis of the disease." Before this opinion can be accepted, microscopic evidence must be adduced to show that the base and margins are not the seat of tubercular infiltration, which Rokitansky affirms is the case. The mesenteric glands are enlarged by the special deposit, and often to considerable size. This, however, it may be stated, is not the essential circumstance in the disease formerly called *tabes mesenterica*. Tuberculous ulcers heal by a

cessation of the morbid deposit taking place, and subsequent effusion of plastic fluid, which becomes organized into the cicatrix tissue before described. The contraction of this, if the ulcer has been of a large size, or extended round the intestine, may cause more or less contraction of its canal. Tuberculous disease generally, but not always, selects the lower part of the ileum as the seat of its chief ravages; it oftener descends, we think, to the cœcum and colon than extends in the upward direction.

Cancer not uncommonly attacks the intestinal canal; it is far more frequent in the large than in the small intestines; out of three hundred and seventy-eight fatal cases from this cause, in two hundred and twenty-one the disease was located in the rectum. All the three species occur in this situation, and colloid more frequently than in most others. Dr. Walshe seems to consider the small intestines to be more frequently affected than Rokitansky does; the latter says that "the colon is almost exclusively the seat of cancerous degeneration," while "the small intestine is scarcely ever the primary seat of cancer, except in the case of acute and extensive encephaloid disease. In the Report of the Pathological Society for 1847–48, there is recorded one case of cancer (colloid) of the small intestines and mesentery, and four of the cœcum, colon, and rectum. The duodenum and upper part of the jejunum are the parts of the small intestine most frequently affected, the rectum and sigmoid flexure those of the large which most frequently suffer. Rokitansky describes primary cancer of the intestines as occurring (1) as encephaloid infiltration of the submucous tissue and patches of Peyer; (2) as carcinomatous infiltration of erectile tissue; (3) "more frequently in the submucous cellular tissue, as round nodulated accumulations; (4) most commonly as an annular deposit of the cancerous tissue in the submucous layer." Mr. Curling has met with an instance of epithelial cancer in the coats of the intestine. When the disease has its seat in the rectum, it most usually occurs at from two to three inches above the anus, according to Dr. Walshe, and tends to spread upwards rather than downwards. When the growth assumes an annular form, encircling the intestine, it produces gradually increasing constriction, which may advance to such an extent that the canal is reduced to the diameter of a goose or even a crowquill. A tuberiform growth, occupying only one side of the intestine, also gives rise to considerable constriction; but this is not so great generally as in the former case. The narrowing of the passage, it is manifest, will be greatest while the growth remains in its original (crude) state; but if, as not unfrequently happens, sloughing and ulceration take place in the morbid mass, the obstructed passage will be again more or less reopened. In many cases, however, the obstruction to the passage of the contents is such that the portion of the canal above the structure becomes immensely dilated, with its muscular tunic much hypertrophied, and its mucous, on the contrary, sometimes thinned, while that below contracts upon itself and becómes very small. Death often takes place in cases of intestinal cancer from the supervention of ileus and inflammation. The ileus (or anti-peristaltic action) is set up not only in consequence of the stricture preventing the passage of the contents of the bowel, for it may ensue when the canal is still

tolerably pervious; but from the paralyzed condition of the walls of the intestine in the dilated and distended part, and from the masses of fecal matter which accumulate there. Inflammation attacks first the dilated part of intestine, "and is there most intense. This portion is discolored, of a dark blue or reddish aspect, its coats are infiltrated with blood; the serous lining covered with exudation is easily detached, the muscular coat is discolored and friable; the mucous membrane, owing to its distension, is devoid of plicæ, villi, or follicles; dark red, distended at some parts with coagula, and sloughy." Sometimes perforation and its consequences take place.

We shall lastly notice some morbid conditions of particular parts of the large intestine. The frequent occurrence of catarrhal inflammation in the cœcum has been already mentioned; but Rokitansky directs attention to a particular form which he terms *Typhlitis stercoralis*, indicating thereby its production by accumulation of indigestible and fecal matters in this situation. Sedentary habits and rheumatism of the muscular coat of the bowels are also stated to be causes of the disease. We believe the proximate cause to be atony of the contractile fibres. "Removal of the accumulated pus, and avoidance of fresh accumulations, generally suffice to establish a cure. If this is not effected, ulcerative destruction of the mucous membrane, and continued sinuous suppuration of the muscular coat, result. In this manner, rapid perforation of the intestinal parietes, and especially of the posterior side, may follow, either inducing extensive inflammation, ichorous destruction of the cellular tissue in the iliac and lumbar regions, and death; or giving rise to general peritonitis," from transmission of the inflammation to the serous membrane. Inflammation sometimes attacks the lax cellular tissue which lies between the posterior surface of the cœcum and the iliac fascia, and is apt to pass into suppuration. In many instances it is doubtless set up by pre-existing, perhaps chronic, inflammatory disease of the bowels; but in others it occurs idiopathically, or metastatically, as Rokitansky states. A calculus descending along the ureter has given rise to abscess in this situation. The purulent matter diffuses itself often for some considerable distance beneath the serous membrane; it has been known to make its way up as high as the kidney, and as low as the interspace beneath the rectum and bladder. The abscess often opens into the cœcum, and also externally. When the catarrhal affection of the cœcum exists in a chronic form, it may cause the condensation of the surrounding cellular tissue, and shrivelling of the intestine itself, so that it "is found converted into a slate-colored capsule, with dense parietes, the size of a walnut or of a pigeon's egg." The cavity of the *vermif. appendix* often affords lodgement to indurated pellets of fecal matter, cherry-stones, or other such bodies, which cause irritation and inflammation, thickening of its coats, and subsequent ulceration. If the irritating substance can be got rid of, and the ulceration ceases, the appendix shrivels up entirely, or in part, according to the extent of the mischief, assuming a lead or slate color. In two very interesting cases recorded together in the Report of the Pathological Society for 1847–48, the vermiform appendix protruded as a hernia, and had undergone ulceration in this situation. In the first, after an

abscess and sinus had been laid open, a small piece of bone, of triangular shape, and with sharp angles, was discharged, and recovery very quickly took place. Death occurring from a different cause not long after, it was found that the appendix vermif. was lying in the inguinal canal; it was enlarged to three times its usual size, its coats much thickened, and its apex opaque, contracted, and adherent to the bottom of the canal. In the second case, there was a swelling of the scrotum caused by the hernial protrusion of omentum enveloping the appendix vermif. Abscesses and sinuses formed in the part, healthy pus was at first discharged, afterwards sanious and offensive matter; the quantity of discharge was profuse, but varied in quantity, and was frequently of a pale orange color. Death occurred from exhaustion, and it was found that the appendix, healthy in structure all the way down to near its blind extremity, was ulcerated at about half an inch from this point, and that a communication existed between its interior and the sinuses of the scrotum. More often inflammation, excited in the vermiform appendix by the presence of hard bodies, extends to the peritoneum, and either at once induces general peritonitis, or gives rise to adhesions, which even if gangrene of the part and perforation occur, may prevent the fatal result for some time. Rokitansky mentions, a curious accident of a different kind which sometimes befalls the appendix. Its canal gets blocked up at a certain part by a foreign body without ulceration taking place. In consequence of this, the mucous secretion accumulates in the closed receptacle, which it distends into a kind of dropsical pouch lined by a thin, serous-like membrane.

The defective state of development of the rectum already alluded to as *atresia ani* is of various degrees, consisting either in simple closure of the anus by the integument being continued across it, or in the rectum terminating in a blind pouch at a greater or less distance from the anus. Sometimes the canal extends for an inch or two upwards from the anus, and then terminates. It is important to remark that when the deficiency in the rectum is considerable, the pelvis is also imperfectly developed, especially in its antero-posterior diameter.

Lacerations of the rectum and anus occasionally take place, all the coats sometimes being torn through, as after a severe labor when the perineum has quite given way, or only the mucous lining being injured, as sometimes happens after the passage of concretions, or hardened feces. The rectum may be excessively distended by fecal accumulations, especially in persons of lax fibre and low nervous power, or when paraplegia exists. Sometimes its channel is much narrowed by the pressure of surrounding organs, when displaced or diseased; a retroverted uterus, an enlarged prostate, a vesical calculus, or a pessary in the vagina, may all have this effect. Rokitansky asserts that hypertrophy of the sphincter ani may give rise to obstinate constipation, and even to ileus, and that it frequently induces excoriation of the mucous membrane, the so-called fissure of the rectum. We think the converse is generally, if not always, the case, that excoriation or cracking of the mucous membrane, by the irritation which it excites, becomes the cause of excessive action and consequent hypertrophy of the sphincter. This is the opinion also of Sir B. Brodie, who says that "the contraction of

the sphincter appears at first merely spasmodic; but in proportion as this muscle is called into action it increases in bulk; and after the affection has continued for some time, it becomes considerably larger." Fissures may be situated at various points, as described by Dupuytren; some, which are below the sphincter, and scarcely involve any texture but the skin, occasion only pruritus. Those which are above the sphincter give to the finger the sensation of a knotty hard cord, and during the act of defecation give rise to indescribable tenesmus. They are commonly produced by the ulceration of internal piles, and mark their situation on the cylinder of feces by a streak of puriform, sometimes bloody mucus. Fissures situated on a level with the sphincter are the worst, being attended with such agonizing pain during defecation, that patients have been known nearly to starve themselves to avoid the recurrence of the action as much as possible. The appearance of these ulcers is that of a narrow fissure, "the bottom of which is red, and the margin somewhat swollen and callous." "Catarrh and blennorrhœa," says Rokitansky, "accompanied by hypertrophy of the coats, which frequently gives rise to plicated and polypous excrescences of the mucous membrane, are very frequent affections of the rectum." Dr. Copland describes *rectal polypi* as varying from the size of a pea to that of an egg, having a broad or a very narrow pedicle, situated high up or low down, presenting generally a mucous aspect, a pale-reddish hue, and a smooth or lobulated surface. A small growth of this kind, which we had the opportunity of examining through the kindness of Mr. I. B. Brown, had a short pedicle, was of the size of a pea, rather highly vascular, of lobulated aspect. It consisted entirely of Lieberkühn follicles, and of

Fig. 225.

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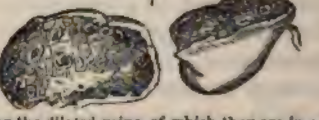


Piles, after excision, showing the dilated veins, of which they are in a great measure composed.

low folds or ridges covered with well-marked columnar epithelium, and mingled with only a small quantity of fibroid tissue. *Hæmorrhoids* depend essentially on a dilated condition of the veins of the rectal mucous membrane, and are quite analogous to the varices of the legs, which are so common. They are named *internal*, or *external*, according as they are situated above or below the sphincter. Although all take their origin in dilatation of the hæmorrhoidal veins, yet in their subsequent progress they come to present different appearances, which we proceed to notice. The first variety, sometimes termed *mariscæ*, are described by Dr. Copland as "fleshy tubercles, of a brownish or pale-red color, situate within the anus, or descending from the rectum. They have a somewhat solid or spongy feel; and when divided they present a compact, or porous and bloody surface. As the blood oozes from the cut surfaces, they become pale and flaccid." Whether internal or external, they often contain a central cavity filled with fluid, or coagulated blood, of a dark color. "More frequently, there is no regular

cavity, the substance of the tumor being as if infiltrated with blood, which becomes coagulated and dark; but this appearance is not owing to extravasation, but rather to a dilatation of a number of small vessels which traverse the tissue in the direction of the axis of the rectum; as, upon dividing the part longitudinally, numerous dark streaks are seen in its substance, while a section made transversely shows only small roundish specks." These tumors elongate, assuming a conical form with bases larger than their necks. Sometimes blood is exhaled from their surface, sometimes only a serous fluid, and sometimes, when they are external, they are quite dry. At first, they generally disappear in two, three, or four days; but return again at an uncertain, or at a regular period, and increase in size, becoming firmer in texture. "After some blood is evacuated from them, or after the determination of blood to the parts has ceased, they collapse, leaving small pendulous flaps of skin, which ultimately disappear if the tumors have been small; but if they have been large, these flaps continue conspicuous, and give a projecting and irregular margin to the anus." Having been strangulated by the sphincter, or repeatedly engorged with blood, or chronically inflamed, these tumors become more permanent and solid. "The permanent state of the tumors is owing partly to the development of capillary vessels, and partly to the effused blood and lymph becoming organized; this latter circumstance especially giving rise to the excrescences, or irregular mass of tumors found around the anus in those subject to hæmorrhoids." The *second variety* of hæmorrhoidal tumors includes such as are formed by a pure dilatation, or varicose state of the veins of the part. Dr. Copland, from whom we continue to quote, describes them "as not so disposed to enlarge at particular periods, and as more permanent, and less painful, than" the first variety. "They are commonly of a dark or bluish color, and soft and elastic to the touch." They are easily emptied by compression, but quickly fill again. "They are round and broad at the base, and often distributed in irregular or ill-defined clusters," which extend often for some way up the rectum, sometimes even as far as the colon. "M. Begin observes that, in most cases, the dilated, superficial, submucous, or subcutaneous veins are only the smaller part of those

Fig. 226.

A slightly lobulated tumor divided in its middle, and the cut surfaces exposed. It was passed per anum. It seems to have been formed by exudation taking place around varicose dilatations of the veins. The cavities seem to have resulted from the dilatation of mucous follicles.

surrounding the rectum. Sometimes the lower part of this intestine appears as if plunged in the middle of a network of dilated and engorged veins, forming a thick vascular ring, the incision or puncture of which may give rise to dangerous hemorrhages." If, in consequence of in-

flammation on the congestion of the varicose vessels, exudations of plastic matter take place around or in the substance of these tumors, they become more solid, and more or less similar to those of the first variety. Sometimes the products of inflammation are deposited within the dilated vessel, which induces its obliteration, and the atrophy of the tumor. Perhaps in some cases the reverse takes place, a vein within an originally solid tumor may become considerably dilated. In short, the varieties met with seem chiefly to depend on the predominance of vascular dilatation, or surrounding plastic exudation. A *third* variety of hæmorrhoidal tumors are described as of an erectile character. They are soft and spongy, and of a purplish color, and give rise to considerable losses of blood. Dr. Colles found in one case "bloodvessels of the size of crowquills, running for some way down the intestine, then dividing each into numerous ramifications, and each forming, by the interlacing of its numerous branches, one of these erectile or vascular tumors. The trunks and branches of these vessels were covered only by the lining membrane of the intestine." Some hæmorrhoidal tumors appear to result from the effusion and coagulation of blood in the surrounding tissue. The reality of this occurrence is denied by Rokitansky, but we quite acquiesce in the remarks of Mr. H. Lee,[1] that it is by no means easy to determine whether the delicate, smooth, and shining membrane lining the cavities in which the coagula are contained, is the lining membrane of the venous system, or one of new formation derived from the blood itself. He also notices the effect of hæmorrhoidal tumors on the mucous membrane around them, which is raised and forced down along with them when they are protruded beyond the sphincter, so that at last it becomes permanently relaxed and "baggy." The female sex, sedentary occupations, and constipation, are enumerated as the chief cause of hæmorrhoids, to which we should add a plethoric habit, and a lax condition of fibre. It does not appear at all proved that cirrhosis of the liver, or obstructive disease of the heart, has any marked influence in the production of piles, as one would naturally expect. The mucous membrane thinned over an hæmorrhoidal tumor is prone to ulcerate, and the resulting sore, according to Rokitansky, is characterized by its seat in the vicinity of the sphincters, its irregular shape, its indented and sinuous flabby margin of mucous membrane, and the ridges of similar tissue that surround or pass over it. These ulcers may continue to burrow into the surrounding areolar tissue, and give rise there to abscess, and ultimately to *fistula in ano*. This, however, more often results from inflammation being set up in the deeper-seated tissues, the areolar and adipose, but still in the vicinity of the bowel, which advances to suppuration, and in most cases makes its way first outwardly through the integument surrounding the anus, and afterwards establishes a communication with the cavity of the rectum by a small aperture situated very constantly at the distance of an inch, or an inch and a quarter, from the anus. It does not clearly appear why fistula in ano should occur so often as it does in persons prone to pulmonary tuberculosis, and still less why its existence should be preservative, at

[1] Medical Gazette, August, 1848.

least in not a few cases, against the invasion of the dread malady. *Cancerous disease* attacks the rectum in most of the forms mentioned as affecting the whole intestine; the only one which it seems desirable especially to notice, is that which gives rise to the annular structure. This occurs almost exclusively at the upper portion of the rectum, especially at the junction of the sigmoid flexure; the strictured part is sometimes unattached, more often firmly agglutinated to the promontory of the sacrum, but is, nevertheless, pushed down by the feculent accumulations above, so as to be within reach of examination by the finger.

VII. ABNORMAL CONDITIONS OF THE INTESTINAL CONTENTS.

The secretion of an excessive quantity of *gas* from the lining membrane of the intestinal canal constitutes the most ordinary form of Tympanitis. It often occurs in inflammatory affections of the canal, which induce more or less paralysis of the coats, in consequence of which the gas is not expelled, but goes on accumulating. The gas in the stomach of an executed criminal was found by Magendie and Chevreul to consist of atmospheric air with a part of its oxygen replaced by carbonic acid, and some hydrogen. In cancerous strictures of the pylorus, and in chronic catarrhal states, the gas contains but little oxygen, much carbonic acid, probably also hydrogen, and carburetted hydrogen, and constantly also sulphuretted hydrogen. In the small intestines of criminals, Magendie and Chevreul found an abundance of hydrogen and carbonic acid, no oxygen, and a varying quantity of nitrogen. Marchand found in the gaseous contents of the large intestines carbonic acid, nitrogen, hydrogen, carburetted hydrogen, and a small proportion in one case of sulphuretted hydrogen. That these gases are secreted by the mucous lining, and do not proceed from decomposition of the ingesta, is considered improbable by Lehmann. We cannot, however, coincide in his opinion, at least to the exclusion of the first-mentioned way of production, if for no other reasons, on account of the experiment performed by Frerichs, which he himself details, that a portion of intestine emptied of its contents, and isolated from the rest of the canal by two ligatures, always became full of gas after being left some time. *Mucus* in any appreciable quantity can scarcely be said to exist in perfectly healthy intestines; but it is secreted abundantly, as we have seen, under catarrhal irritation. Rokitansky applies to it the following epithets in various cases; it is either milky white, yellowish, and purulent, or glutinous, transparent, vitreous, spawny. He also states "that there can be no doubt that a peculiar gelatinous constitution of the mucus is the nidus of intestinal entozoa, and the cause of helminthiasis." Under irritation of an acute character small membranous patches of mucus are often passed; we have seen these in a case of dysentery constituting the whole of the scanty evacuation. Between these and the fibrinous tubular formation before noticed, there is no very essential difference, and both are to be distinguished from certain membranous substances which occasionally appear in the evacuations, and may cause

some perplexity to the patient and physician, unless their nature be understood. These are simply the undigested remains of some tendinous expansions, which contain a great deal of yellow elastic fibre, on which the gastric juice seems to act with much less energy than on the white. Rokitansky says that "the occurrence of an excessive elimination of feces from the intestinal secretions is an established fact. It takes place as a critical discharge in various diseases," but may also occur as an idiopathic affection, "which may, by the excessive drain it causes, give rise to atrophy of the intestinal coats, and to general emaciation.

Concretions are occasionally met with in the intestinal canal, and in rare instances of very large size. Dr. Monro (Primus) observed some varying from five to eight inches in circumference, and Monro (Secundus) removed one from the colon which weighed four pounds. Sometimes several, as many as thirty, exist together, but commonly there are not more than two. The color of the smaller resembles that of iron ochre, the larger are more of a coffee color, and occasionally whitish. "They are generally found in concentric layers, and are often radiated, sometimes very obscurely from nuclei. They are more or less porous, either spheroidal or oblong, and vary from the size of a pea to that of a hen's egg, or still larger." The nuclei of concretions may be gall-stones, fragments of bones, fruit, seed, &c., round which saline and animal or undigested matters collect and become condensed. In Scotland, where oatmeal is much used as an article of food, the fibres of the husk of the oats have been found to constitute a large part, or nearly the whole of the concretion; and in a similar manner, chewed paper, the several portions being matted together by mucus with fecal and earthy matter, has been known to cause their formation. Such concretions often exhibit no distinct nucleus. Concretions, which may be mistaken for gall-stones, but which are of a fatty nature, are sometimes voided by persons who suffer from a torpid state of the bowels, and deficient digestive function. Dr. Copland describes them as of a globular form, varying from the size of a pea to that of a large grape, of a cream color, slightly translucent, and of the consistence of soft wax. It is probable that these concretions, as well as the evacuations of a more fluid fat which occasionally take place, are connected in some measure with disease of the pancreas. Observation of disease has in several instances shown the coincidence of the two phenomena; and Bernard has offered a physiological explanation, viz: that the use of the pancreatic secretion is to make the fat contained in the food capable of being absorbed. His results, however, are denied by Frerichs, Bidder, and Schmidt. *Blood* is sometimes effused in greater or smaller quantities into the intestinal canal. This may result from active or passive hyperæmia, ulcerations, purpura, scorbutic dysentery, and we have once seen it in a case of death from a severe fall.

CHAPTER XXXIII.

ABNORMAL CONDITIONS OF THE LIVER.

Congenital malformations are rare; absence of the liver is only observed in extreme cases. Sometimes the left lobe retains, in a greater or less degree, its fœtal proportion to the right.

Congestion. The vascular apparatus of the liver is very large, its capillaries are more capacious than those of most other parts, and the caliber of the portal, and especially of the hepatic veins is extremely ample. It is, therefore, capable of containing a very large quantity of blood. Though its surface is closely invested by its capsule, yet this membrane allows of a good deal of distension, and this is also attested by the tortuosities of the arterioles of the surface, which probably exist for the purpose of allowing the vessels to be elongated without injury to their texture. Congestion of the liver may be general or partial. The latter is far the more common condition, and, indeed, in its lower degrees is not morbid. The central parts of the lobules, in perfectly healthy livers, are often seen to be marked by a spot of redness which occupies about the middle two-fourths of the whole diameter. This is produced by the blood, as the circulation gradually failed, having stagnated in the hepatic veins, in their radicles, the intra-lobular veins, and the surrounding capillaries. Mr. Kiernan named this, "Hepatic Venous Congestion of the first degree." When the congestion extends further in the direction backwards, there are seen no longer mere spots of redness, but patches of very irregular shape surrounding, more or less completely, portions that are not congested. These portions are situated at the interlobular spaces, where three or more lobules adjoin. Such a condition was named by Mr. Kiernan, "Hepatic Venous Congestion of the second degree." This may coexist with a perfectly healthy condition of the cells which occupy the meshes of the capillary plexus, but not unfrequently these are variously altered, and thus give rise to some modification of the appearance.

The "*nutmeg*" condition may be here referred to; it consists, in its best-marked instances, of deep red congestion, forming patches and streaks, occupying the central parts of the lobules, and surrounded by patches of a grayish, or dirty white color. The congested portions are most definitely limited, and the contrast between them and the pale parts is extremely striking. This probably depends on the circumstance that the portions thus devoid of blood are affected with fatty degeneration, a change which, by causing the cells to increase in bulk, occasions compression of the interwoven capillaries. That this is the true cause of

the limitation of the congestion is proved by its exactly ceasing at the inner margin of the zone of fatty degeneration. Many of the cells in the congested part are seen filled with dark yellow matter; very many,

Fig. 227.

Section of liver, showing the nutmeg appearance; the dark parts are the deep red congested central parts of the lobules.

also, are atrophied, probably in consequence of the pressure exerted by the distended capillaries. The nutmeg appearance may be exhibited in some degree, by livers which are quite free from fatty degeneration, but it is never so marked as in the condition just described.

When the congestion extends still further and becomes general, occupying every part of the liver, the organ presents a deep red color

Fig. 228.

(A) Section showing lobules of the liver, bounded by marginal zones in a state of fatty degeneration. The interior of the lobules is deeply congested, and contains much dark yellow pigment in masses.

(B) Cells loaded with pigment, atrophied cells and granular matter from the interior of the lobules.

throughout, though, even in this case, the centres of the lobules present the darkest tint; it is also enlarged often to a greatly increased size,

and becomes more firm and prominent, so as to be readily perceptible to the touch, below the margin of the ribs, on the right side. The result of injecting a liver which is nearly drained of its blood, as one taken from a slaughtered sheep, is very instructive as to the amount of enlargement that may be produced by congestion. The organ swells up as the fluid is thrown in, and when fully injected, is of nearly double its former size, greatly more dense and solid, with its thin anterior margin prominent and hard. Such a state may well produce a sense of weight and fulness in the right hypochondrium.

The most frequent cause of congestion to any degree that can be considered morbid is organic disease of the heart, especially such as produces great obstruction to the circulation, and throws the blood back upon the right side of the heart, and the venous system in general. All causes of apnœa produce the same effect, and act in the same way. Congestion thus induced is passive; active congestion takes place in inflammation of the liver, or, as we have observed, when large doses of calomel are administered. The congestion which occurs in the cold stage of ague and other fevers, seems to depend simply on the recession of the blood from the surface, and is, therefore, more of the nature of passive than of active.

Portal venous congestion is a rare variety of partial; the centres of the lobules are pale, and are surrounded by continuous red zones. It is said to occur in children only.

Congestion of the liver, although extreme, does not seem to occasion any structural change, if it is only temporary; but if, as in the case of obstructive cardiac disease, it results from a permanent cause, and is, consequently, itself permanent, it produces the following effects: The distended capillaries of the portal-hepatic plexus, press on the intervening cells; these become, in part, atrophied or stunted, in extreme cases almost destroyed: in part, they are gorged with yellow matter to such a degree that they appear as opaque masses. The quantity of yellow matter thus formed is far greater than any that exists in healthy states of the organ, and, as some of it is doubtless absorbed and carried into the blood, we find in this circumstance some explanation of the icteric hue which is so often observed in such patients. The connection which certainly exists between the congestion and the yellow engorgement of the cells, as cause and effect, gives additional support to the opinion that the yellow bile-pigment is a modification of, and derived from, the coloring matter of the blood. Whether long-continued congestion produces still further changes, is not yet made out clearly. We have often thought that the lobular parenchyma was infiltrated by a dense, homogeneous, solidified blastema, distinct from the debris of the atrophied cells, but this may have resulted from a different cause.

Hemorrhagic effusion may take place as the result of extreme congestion, but this is rare; the blood may be poured out either on the surface, detaching the capsule for some extent, or deeper in the substance of the organ. The former occurrence, Rokitansky states, is most common in infants, and may even proceed to such an extent as to rupture the serous investment, and allow the escape of blood into the peritoneal cavity. The deep-seated extravasations occur more frequently in adults

than the superficial, and constitute apoplectic spots of various forms and size. After their partial absorption, a cellulo-fibrous cicatrix remains.

Inflammation of the liver must be considered as affecting either the substance of the organ, chiefly, or its capsule. The former is, in temperate climates, a rather rare affection, the latter extremely common. Acute inflammation of the parenchyma of the liver produces general congestion and more or less softening. "These effects," Rokitansky says, "are confined to one or more patches; the congestion, though general, is not universal. The viscus is swollen in proportion to the number and size of the inflammatory patches, and this tumefaction is particularly perceptible when a section is made, the turgid tissue rising above the edges of the incision and the peritoneal sheath." The parenchyma "is loosened and lacerable;" this depends in great part on the cells losing their natural cohesion together, so that they no longer form radiating series. In the latter stages of inflammation, the red color of active congestion fades, and is replaced by a brownish or grayish-red tint in some parts, with yellowish-red or pale-yellow in others. Abscess is a frequent result of acute inflammation of the liver: it is sometimes preceded by a short stage of diffuse suppuration, when the form of the lobules can still be recognized, though their substance is very soft and of a yellowish color. The commencing abscesses are at first very small, like spots of purulent matter dispersed here and there through the inflamed and softened tissue; they gradually enlarge, several coalesce together, and thus form cavities of irregular shape and size. The parietes of the abscess are uneven, presenting the remains of former partitions; they are covered by a kind of pyogenic membrane, which consists, in great part, of pus-globules; external to this, the wall is formed by hepatic tissue, infiltrated with exudation-matter, which serves to bound and limit the abscess, as in the case of a common phlegmon. In very small abscesses, and those of very recent formation, as also in cases where the vital powers are greatly depressed, this limitation of the abscess by exudation-substance does not take place; on the other hand, in old abscesses, and those of very large size, occurring in tolerably healthy systems, a strong enveloping cyst may be found, consisting of fibroid tissue, and amounting sometimes to three or four lines in thickness. If the abscess be deep-seated and encysted, it may continue, especially if of small size, for a long time, without increasing much, or producing serious disturbance of the health; but, if it be near the surface, it excites inflammation of the serous investment, and effusion of lymph, which soon unites its wall with the part with which it is in contact. This may be the wall of the abdomen, or some of its contained hollow viscera, or the diaphragm which roofs it in above; and by any of these various routes the abscess may extend, and at length discharge its contents. By extension, also, in the parenchyma of the liver, the abscess may reach a branch of the portal vein or of the hepatic, excite inflammation of its coats, and consequent obstruction of its canal by exuded fibrin. When the enlarging abscess reaches an hepatic duct-branch, it affects it in a different way; it does not set up inflammation in its walls, and cause its obstruction (though this may no doubt occur in some of the smaller), but it ulcerates through its tunic, and establishes a commu-

nication between the efferent channel and its own cavity. Hence, it occurs that the pus contained in large abscesses is always mingled with a considerable quantity of bile, while that of the smaller and recent abscess is almost pure. An abscess of the liver may sometimes heal either after the evacuation of its contents, or after they have been absorbed; the latter occurrence is, we should conceive, exceedingly rare. In either case, the walls of the abscess approach each other, and at last collapse together, including sometimes a quantity of solidified purulent matter, which at a later period forms a cheesy or cretaceous mass, attesting, by its presence in the midst of the cicatrix, the nature of the changes which had previously taken place.

Several different causes have been assigned for the formation of abscess in the liver, but none are probably nearly so effective as that to which Dr. Budd has especially drawn attention. Dysenteric ulceration, or any accident producing inflammation of the veins that originate the V. Portæ, charges the current of blood proceeding to that organ with puriform matter, which, in its passage through the capillaries of the lobules sets up secondary inflammation, attended with albuminous exudation, and terminating quickly in the formation of matter. It is not clearly made out in what way injuries of the brain act in producing abscess of the liver; but it is probably in the same way, though by a less direct channel than that just described; so, also, many regard the occurrence of the so-called secondary depôts in the liver, subsequent to amputations, and other great operations, as the result of phlebitis attacking the veins of the injured part. The only other causes which can be named with any certainty as producing abscess are, external violence inflicted on the region of the liver, and violent acute inflammation of the organ, such as occurs in tropical climates. It is worthy of remark, that typhoid and tuberculous ulcerations of the intestines, as well as those consequent on burns, do not, at least, have not yet been observed to, produce abscess of the liver.

The capsule of the liver is very often indeed attacked with inflammation, or presents, on post-mortem examination, such changes as are usually ascribed to this process. Bands of adhesion of various length, and attaching different parts of its surface to contiguous organs, are very often found: these are pretty certain evidences of an acute or subacute by-past hepatitis; they are often traversed by newly-formed vessels which establish a communication between the capsular arterioles and the contiguous vessels of the general system. In a specimen which we injected, the vessels of a patch of false membrane, on the surface of the capsule, are seen to be of much greater caliber than the vessels in the adjacent healthy part. Adhesions commonly form, as said, over superficial abscesses of the liver: it is rare that this fails to take place, and that an abscess bursts into the peritoneum. Over hydatid tumors and cancerous masses they are less frequently formed. Dr. Budd particularly notices their absence, and considers that their production is the exception and not the rule. This may, perhaps, be in some measure accounted for by another circumstance relative to cancerous tumors in this organ, mentioned by the same observer, viz: that they seem capable of tainting, with their own peculiar morbid action, opposed parts with

which they come in contact; such tainting of a part may be conceived to be quite inconsistent with the effusion of comparatively healthy lymph. The capsule of the liver is very frequently found thickened, appearing whiter, more opaque, and dense; this thickening generally occurs in patches of various size, which sometimes coexist together with the bands of adhesion just noticed, but quite as often are independent of them, or coincident with similar thickenings of other serous membranes. It seems doubtful whether these changes should not be more properly classed as degenerations than as inflammations.

Gangrene of the liver is very rare; Rokitansky well observes that "it is developed in parts affected with inflammation and suppuration, not so much as a result of intense inflammation as of certain peculiar conditions which cause a tendency to gangrenous degeneration." These peculiar conditions may be probably either low, unhealthy states of the general system, or, as in the interesting case recorded by Dr. Budd, the septic influence of a previously healthy part which had been affected by gangrene.

Phlebitis of the portal or hepatic vein-branches has already been alluded to as resulting occasionally from abscess of the liver, but it may be produced by other causes. A fish-bone has been known to perforate the wall of the stomach and the head of the pancreas, and wound the superior mesenteric vein, exciting thus inflammation of the coats of the vessel which extended to the divisions of the portal vein. The effects of phlebitis are the same in these as in other veins; it may simply occasion the effusion of lymph and blocking up of the channel with fibrinous coagula, or it may also proceed in some portions of the vein to the formation of pus.[1] In the former case, when the inflammation has subsided, the obstructed vessel gradually shrinks, and is reduced to a fibrous cord, while the surrounding tissue to which it was distributed, being deprived of its supply of blood, atrophies and falls in, so that the course of the vein is indicated by a deep linear fissure. If suppuration occurs, a string of small abscesses is produced in the tract of the obliterated vein, the abscesses being connected together by a dense fibroid cord. The consequence of such attacks must be the diminution, to a greater or less extent, of efficient hepatic parenchyma, and as the importance of this structure to the due performance of nutritive absorption is very manifest (were it attested only by the intimate relation in which it is placed with the portal blood), it seems very probable that the abiding emaciated and enfeebled condition of many persons who have suffered inflammatory attacks of the liver, really depends on their having been thus deprived of a greater or less part of this important organ.

Cirrhosis.—A liver which is affected by this change in an extreme degree is remarkably altered. It is much smaller than natural, much

[1] In a case occurring at St. Mary's Hospital, all the portal veins throughout the liver, up to those of a very small size, were converted into channels with ragged walls filled with a tenacious pus. Here and there the vein-channel was enlarged into an irregular pouch, but there were no distinct abscesses. The cause of the phlebitis appeared to have been a patch of ragged ulceration at the origin of the portal vein. The parenchyma was not atrophied. The hepatic ducts were not inflamed, but the gall-bladder and duodenum appeared so.

paler, and instead of presenting a smooth surface, is contracted and puckered so as to resemble, according to a former observer, "a conge-

Fig. 229.

Section of liver in a fatty state, with abundant new-formed fibrous tissue between the lobules.

ries of little firm globules like the vitellarium of a laying hen." These globular portions are of various sizes, and evidently consist of parenchymatous substance; they are surrounded, and as it were capsulated by firm fibroid tissue which extends throughout the whole liver, and gives it a remarkable degree of density and firmness. This fibroid tissue is evidently a new formation, and as such tissue frequently does, it contracts and shrinks together, and so draws in the surface at various parts as to produce the irregular nodulated, or "hobnail" condition, as it is familiarly termed. The same shrinking also affects the vessels which supply the liver; they are surrounded and ensheathed by fibrous tissue in the healthy state, and when this is morbidly thickened and condensed, the pressure exerted upon them narrows their channels and materially diminishes the quantity of blood which they are able to convey. Hence the portal current is checked at its very origin, and the congested capillaries are obliged to relieve themselves by effusion of serum into the peritoneal cavity. The capsule of a cirrhotic liver is sometimes smooth, sometimes thickened or attached by adhesions to adjacent parts; these adhesions are often traversed by newly-formed vessels, which form a kind of collateral circulation between the portal vein and the general system.

Such is a brief description of a liver affected with ordinary cirrhosis; but there are other conditions of the organ, essentially similar, in which the external appearances are very different. The organ is firm and dense, evidently from the presence of an increased quantity of fibroid tissue; but its surface is not puckered, or but slightly, nor its edges rounded; instead of being pale, it may be highly congested with blood, usually of the hepatic venous second degree, this often depending on obstructive cardiac disease. Microscopic examination shows that the quantity of fibroid tissue forming the Glissonian sheaths is considerably increased, sometimes to such a degree as to encroach on the lobules to

a great extent, and produce atrophy of their substance. It is possible that some livers which present these appearances may be in an early stage of cirrhosis, and would subsequently become contracted and nodulated; but we strongly incline to the belief that this is not the case

Fig. 230.

Fibres originating from nuclei from fibrous tissue of a cirrhotic liver. Some cells are figured of the natural size, others are very much atrophied.

with the majority, and that the morbid alteration is somewhat different. The difference probably consists in the more general and extensive formation of fibroid tissue throughout the liver, in its being less confined to the portal canals. It is rather remarkable that the hepatic cells in extremely contracted cirrhotic livers present a tolerably healthy aspect; their nuclei are distinct, and though, perhaps, containing less oil than usual, they are by no means destroyed or seriously altered. They are far more affected in the dense, firm, uncontracted livers, partly as the result of congestion with impletion of yellow matter, partly by atrophy from the encroaching fibroid tissue. The minute ducts which run in the smaller portal canals and between the lobules are often atrophied by the pressure of the condensed fibrous tissues, so that they can no longer be detected; in this way the biliary secretion may be materially interfered with.

It seems to be proved by the observations of Dr. Bright and Dr. Budd that in the early stage of cirrhosis the liver is enlarged; this would seem to depend on the effusion of lymph and serum within its texture during the existence of inflammatory action. In many cases there can be little doubt that this is the case, and that the cirrhotic change results from a subacute inflammatory action being set up in the Glissonian sheaths. But in many cases we are disposed to think the process is different; that, both in the finally contracted and uncontracted livers, the fibrous tissue is hypertrophied and condensed rather by a degenerative action than by one which can be termed inflammatory. The change seems to be of a similar kind to that which produces cartilaginoid induration of the capsule of the spleen, stiffening of the valves of the heart and contraction of its orifices, which can scarcely be regarded as of inflammatory origin. We are confirmed in this view by having often observed various minor degrees of condensation and thickening of the Glissonian sheaths in cases where there was no trace of inflammatory action, as well as by a circumstance which hitherto has been quite unexplained. This is, that the spleen, albeit exposed to the

backward pressure of the blood retarded in the splenic vein, does not become distended in the way that one would expect, but is often, on the contrary, small and soft. In such spleens, we have often observed very many of the nuclei throwing out fibres, which is certainly not their natural metamorphosis; and hence it seems not improbable that in this way, owing to the increase of fibrous tissue in its substance, the parenchyma of the spleen is less distensible than usual, and has a contrary tendency to shrink and collapse.

The remote cause of cirrhosis, in many cases, is certainly the habit of spirit-drinking: the alcohol absorbed into the portal blood first passes through the liver, and very probably exerts some action on its tissue. This suggests the idea of the spirit acting as a local irritant, and with this Dr. Percy's observation of the greater affinity of alcohol for the liver than for other organs in animals poisoned by it seems to harmonize. But it is most probable that the crasis of the blood is also changed, and that this fluid comes to be in that condition which Rokitansky denominates the fibrinous crasis. This would also account for the similar changes which are often found in other parts coexisting with cirrhosis. Obstructive cardiac disease is probably not a direct cause of cirrhosis in any of its forms or degrees, but certainly must be a predisposing one. Congestion favors the occurrence both of inflammation and of degeneration. Both the heart disease (*e. g.* constriction of the mitral orifice), and the cirrhotic change in the liver, are often, no doubt, common results of the same condition of the blood, viz: that to which we have above alluded. There are, no doubt, other exciting causes besides the ingestion of alcohol, but they can scarcely be particularized; we believe them to be in general such as increase the quantity and alter the quality of the fibrin of the blood.

The next condition of the liver which we shall describe is that of *fatty degeneration*. A liver thus affected is usually much enlarged,

Fig. 231.

Section of liver in an advanced state of fatty degeneration. The cells are much broken up and fused together.

paler than natural, and, in most cases, softer: sometimes, however, it has a feeling of great solidity. The capsule, in cases of uncomplicated

fatty degeneration, is not thickened, nor attached by adhesions to adjacent parts. The thin edge of the organ is somewhat rounded, and the thickness generally increased. On microscopic examination, it is at once seen that the hepatic cells are engorged with oil: instead of containing a few minute drops imbedded in a mass of granulous matter, they are filled to the extent of one-half or two-thirds, or even their whole cavity, with colorless fluid oil. Sometimes a quantity of yellow matter is also seen in the cell-cavity, together with the oil, but this is often absent. The nucleus disappears, as is generally the case in cells that have fulfilled their work of secretion, but the envelop persists, and is sometimes a little thickened and striated. In very advanced cases the cells are not found merely gorged with oil, but, to a great extent, broken up and lost; in their place there are seen only granular debris, entangling multitudes of oil-drops of different sizes. There seems to be less tendency to the development and growth of young cells than in the healthy state; those that are forming appear stunted, and many become very early the seat of oily engorgement. The fatty change is very often confined to the margins of the lobules, and is always, we think, most advanced there; sometimes, however, it may commence in the centres of the lobules. The pale condition of the liver depends on the enlarged size of the cells, which are pressed closer together, and thus constrict the capacious capillaries and allow less blood to be contained within them. There is, however, no obstruction to the flow of blood, such as we have seen in cirrhosis; the soft state of the oil-laden parenchyma sufficiently accounts for this. We think it most probable that fatty degeneration does not consist merely in the impletion of the cells with oil, in their containing a greater quantity of this matter than they naturally would, but that there is an actual impairment of their development and nutrition, of their active power of formation and secretion, so that the tissue really undergoes a kind of decay. In support of this view, we may mention that we have scarcely ever found sugar present in the parenchyma of a thoroughly fatty liver, while it can almost always be detected in livers that are not so affected; this is certainly a remarkable fact, and seems to show that one very important function of the liver in such cases is, at least, very imperfectly discharged. It is an interesting fact, that the bile in these cases undergoes no constant or necessary alteration; it is sometimes unusually pale, but often has the dark green tint of ordinary bile. The minute ducts are tolerably natural, and doubtless continue to discharge their part of the bile-secreting process. A fatty condition may coexist with a considerable degree of cirrhosis. The appearance of a section under the microscope is then very remarkable; the lobules appear as opaque islets

Fig. 232.

(a) Empty envelop of an hepatic cell, from which the oil has escaped.

(b) (c) (d) (e) Hepatic cells containing much oil.

separated more or less widely from each other, by more transparent spaces of fibroid tissue.

There is a variety of the fatty liver which is termed the *waxy*. It is described by Rokitansky as having a color resembling that of beeswax, and being more consistent or firm, dry, and brittle than the ordinary fatty liver; the color probably depends on the presence of yellow bile-pigment in the cells, and the increased firmness and brittleness on the oil being replaced by some of the more solid kinds of fat.

The proximate cause of the production of fatty liver is, we believe, the existence of an undue quantity of oily matter in the blood, in proportion to the assimilative power. In cases of disease, it is also very probable that the vital power of the hepatic cells is much lowered. It may, therefore, occur: (1.) When an animal is largely fed on food containing much fat. (2.) When, in the course of an exhausting disease, rapid emaciation takes place, and causes the blood to become loaded with oil from the waste of the adipose tissue. (3.) When the type of respiration is low, and the blood necessarily, therefore, contains much hydrocarbonous matter. In the first and third class there may be simply accumulation of a large quantity of oil in the tissue of the liver; in the second there is generally, also, more or less degeneration of the hepatic structure. We have verified this by positive observation, as respects the first and second class. Animals fed for some time with fatty food have their livers loaded with oil, but the cells are not at all destroyed; they contain much oil, and much also is deposited between them. We have found the liver, in persons dying, in a condition of extreme emaciation from other diseases besides pulmonary phthisis, in a complete state of fatty degeneration, and this has occurred so often, that we should expect to find it in most cases of great general wasting. It is certainly the emaciation of consumptive disease that produces fatty transformation of the liver, and not the mere destruction of the oxygenating apparatus. This statement is strongly confirmed by the analysis of about a hundred cases, in which we examined, microscopically, the condition of the liver. Among them, there were eighteen in whom the liver was thoroughly fatty, or nearly so; of them, only *one* died of pulmonary phthisis, four others of scrofulous affections, the remainder of diseases having no connection with any form of phthisis; ten cases of phthisis occurring in the same list, presented either no fatty degeneration, or an imperfect and partial change only. A fatty state of the liver is sometimes coincident with a similar condition of the kidneys, but not by any means invariably.

Another form of degeneration of the liver, is that which Rokitansky has termed the *lardaceous*, or *bacony*. This term simply expresses the idea which the appearance of the morbid tissue conveys, and is, perhaps, preferable to that of scrofulous enlargement, which has been given to this form of disease by others, inasmuch as evidence is certainly wanting of the new deposit being identical or similar to any known kind of scrofulous matter. Rokitansky thus enumerates the characteristic features of this condition: "considerable increase of size and weight, with remarkable lateral development and flattening of the organ; smoothness and tenseness of the peritoneal investment; a certain degree of doughy consistency, combined with hardness and elasticity; anæmia; pale, watery,

portal blood; gray, grayish-white, or grayish-red color, tinged with yellow or brown; the surface of a section being smooth and homogeneous, resembling bacon." In thin sections it is well seen, under the microscope, how the normal tissue is infiltrated with, and partly replaced by a homogeneous, refracting substance; this forms small masses of various size, which lie heaped together between the hepatic cells, and so compress and atrophy them, that they form in many parts a kind of wide-meshed plexus, the intervals of the plexiform bands being occupied by the glistening bacony matter. When liq. potassæ is added, this matter loses much of its refracting power, and is reduced to mere delicate films; it shows no trace of organization, and seems to be deposited rather between than in the cells themselves. The cells, in such cases, often contain oily matter, or yellow pigment; the oil remains very apparent after the action of liq. potassæ, and is very distinct indeed from the bacony matter. Deposits of similar matter are not uncommon in the spleen, and we have seen it also in the capillaries of the Malpighian tufts of the kidney and in the gastric mucous membrane. The exact nature of the substance thus deposited is unknown, but it is probably a variety of deteriorated albuminous matter. Neither does it seem to be special to any particular form of disease, but to occur generally in persons whose constitutions are gravely impaired. Rokitansky states it to concur with scrofulous and rickety disease, with syphilitic and mercurial cachexiæ, and occasionally to appear as a sequel of remittent fever in cachectic persons. We have seen it produced in the spleen, as well as Bright's disease of the kidneys, apparently from the injurious drain on the system occasioned by an empyema, discharging externally. The presence of the abnormal matter in the tissue of the lobules causes obstruction to the current of the portal blood; it is thrown back on the capillaries of the intestines, and ascites or sometimes diarrhœa results.

The next morbid condition of the liver which we shall consider is that which exists in the various affections in which *jaundice* forms a prominent symptom. There is little doubt that in most cases a jaundiced condition of the liver precedes and occasions a similar condition of the whole body. General jaundice is commonly supposed to depend upon the absorption into the blood of bile that should have passed out into the intestine, and this is, doubtless, the cause of it in many cases. In these, the bile locked up in the substance of the liver causes it to be tinged yellow, a result which we have produced artificially by placing a ligature on the duct. com. choled. in animals. But in many cases more than this occurs. Dilatation of the heart, or obstructive valvular disease, throwing back the blood on the venous system, occasions permanent congestion of the liver, and often produces the condition termed "nutmeg" in its most marked form. In this, as before described, the congestion is exactly coextensive with extreme yellow engorgement of the cells; a much larger quantity of yellow matter is contained in the liver; there is hepatic jaundice, and together with this, and, no doubt, in consequence of it, general jaundice frequently occurs. In these cases, we think the evacuations continue of their natural color; a certain quantity at least of bile flows into the intestine. In most healthy animals the cells of the liver have only a very faint, if any, yellow tinge,

but, by repeated doses of calomel, we have caused the production of a large quantity of yellow matter in the cells; there is evidence also to show that the same has occurred in the human subject. In the acute yellow atrophy, as Rokitansky names that condition of the liver which is found in cases of jaundice occurring often in several members of a family, one after another, attended with symptoms of toxæmia, and proving fatal by coma, there must certainly be a greatly increased production of yellow pigment. The flow of bile into the intestine is not so completely stopped as it is in other instances of jaundice, and the yellow coloration of the liver is deeper than it is almost ever seen. *Icterus neonatorum* appears also to be an instance of the excessive production of bile or of bile pigment: there is evidently no disease of the liver, or any obstruction in the biliary ducts, but owing to the organ at birth being highly congested with blood, and the system not having adapted itself to its new condition, a greater quantity of yellow pigment is formed out of the coloring matter of the blood than can be readily carried off by the bile; this again returns into the blood and produces jaundice. This explanation seems preferable to that which assigns a kind of hyperæmic, or half-bruised state of the skin, as the cause of the yellow stain; were this so, how could the conjunctiva come to be affected? When jaundice occurs in the course of fevers or in pyæmia, it then depends, in all probability, on an alteration taking place directly in the hæmatin of the blood, which, as in the case of an extravasation, is changed from a red to a yellowish tint; in this case, there would be no preceding jaundice of the liver. It is evident that, in all instances of jaundice, the unnatural tint results from the presence of a yellow (usually identical with bile) pigment in the blood; this is easily demonstrable in the urine and other secretions, by the stain imparted to linen, or by the play of colours which it gives with nitric acid; but there is little or no evidence to show that real *biliary matter* is present in the blood or the secretions. From jaundiced livers plenty of yellow pigment can be extracted, reacting with the nitric acid test, but no cholic acid, or any substance that gives the reaction of Pettenkofer's test. The blood, in cases of jaundice, is more often found to contain bile-pigment without cholic acid, than the reverse. The same is the case with the urine; it often gives a characteristic reaction with nitric acid when none is afforded by the test of sugar and sulphuric acid. From these data, we must conclude that jaundice depends on the presence of bile-pigment, or some similar modification of hæmatin in the blood; but that it is by no means certain that bile, as such, is actually present. It may be that, in the graver cases of jaundice, attended with toxæmia, cholic acid, or some modification of it, is present in the circulation in large quantity, as well as yellow pigment. The color of the liver in jaundice is of a more or less marked yellow, in some cases passing to a green or brownish tint; this will be, of course, modified, and more or less concealed by the blood contained in the vessels. There is no other particular change to be noticed except in the acute yellow atrophy. Here, the color of the organ is an intense yellow; its texture is flabby, a section shows nothing of the natural lobular arrangement; its size is greatly diminished, and it seems almost or quite bloodless. The blood in the large

vessels, is said by Rokitansky to be reduced in consistence, and of a dirty reddish-brown color, and the coats of the V. Portæ to be tinged with bile. Under the microscope, it is seen that the cells are completely destroyed, and even their nuclei have perished; the parenchyma is a mere mass of broken-up granular matter, tinged deeply yellow and containing some largish yellow masses, together with diffused oily matter. In one instance which we examined, no sugar could be extracted from the liver, though it is usually abundant in healthy organs; this may, however, have depended on the non-ingestion of food for some time before death. The minute ducts we have found gravely altered; they had lost their natural structure, and were filled with subgranular matter and opaque whitish globules. These globules, probably a kind of concrete oily matter, were very abundant in the lymphatics, and rendered their course remarkably distinct.

The following may be enumerated as adventitious growths in the liver: (1.) Lipomatous tumors. (2.) Cavernous tissue. (3.) Tubercle. (4.) Hydatids. (5.) Cancer. The *lipomatous tumor* is very rare, and, according to Rokitansky, seldom larger than a pea. *Cavernous tissue* is said by this celebrated pathologist to be of frequent occurrence; we doubt, however, whether this is the case in England. Its size varies from that of a hempseed to that of a hen's egg, or still larger; it is of dark color, from the quantity of blood occupying its cells, and is thus seen through the peritoneum of the surface more or less prominent, according to the degree of distension. If its cells are empty, it is found collapsed and shrunk. These growths may be single or numerous. *Tubercle* is a deposit not very unfrequent in the liver, at least to a small amount, but it is very rare that it forms large masses, or that it undergoes softening or other changes. It most frequently appears in the shape of semi-transparent, grayish, miliary granulations, or of yellow crude tubercle; the masses are of a small size, seldom larger than, or even so large as, a pea; they are generally few in number and widely scattered. We think we have often seen nodules of fibrin deposited in the substance of the liver, which might easily have been confounded with the miliary granulations; their microscopic characters would sufficiently distinguish them. Rokitansky mentions a state of acute tuberculosis as occasionally affecting the liver; it is found then "in a peculiar state of turgescence, the tissue relaxed, friable, pale, and gorged with a serous or sero-sanguineous fluid." Such an occurrence indicates a high degree of tuberculous dyscrasia, "a tendency to universal tubercular deposition, and especially in the abdominal viscera." The tubercle thus tumultuously deposited occasionally softens and breaks down, but not exactly in the way of suppuration that it does in the lungs, and thus an hepatic vomica may be formed, "which offers no peculiar characters beyond the biliary discoloration of its contents." It appears that such a vomica may be occasionally confounded with a condition to be subsequently described, in which a cystic dilatation of the minute biliary ducts takes place, the cavities of the cysts becoming filled with a whitish cheesy matter.

Hydatid cysts are of frequent occurrence in the liver, more so in this than in any other organ. Sometimes they are single, sometimes there

are several separate cysts. They often attain a considerable size. Rokitansky mentions one in the Vienna Museum of a foot in diameter, and we have very recently examined one of an oval form, whose long diameter measured six inches. Their usual site is the right lobe, and the largest are generally found here, but the one just mentioned was situated at the extremity of the left, and had grown in, and far beyond, the left lateral ligament. As their size increases, they rise to the surface of the liver, and sometimes excite inflammation of the serous membrane, by which adhesions are formed connecting them with the parts adjacent. The prominent part is, of course, that where least resistance is offered to the pressure of the fluid within, and its wall may hence atrophy and give way, or be destroyed in the course of suppurative inflammation, and the contents thus be effused into some neighboring cavity. The cysts have been known to burst into the peritoneal sac, into that of the right pleura, or into the bronchi of the corresponding lung, into the duodenum or transverse colon, and in some rare instances, into a large bloodvessel or branch of the hepatic duct. When the tumor, in its progress, causes ulceration of one of the smaller ducts, which is not uncommon, bile makes its way into the cavity, mingles with and tinges its contents, and very often excites suppurative inflammation of the walls of the sac. This seems to be the reason that hydatid tumors in the liver suppurate much more frequently than those in other parts.[1] Other circumstances, however, may certainly cause these cysts to inflame and suppurate. The detailed description of the structure of hydatid cysts will be found under the head of Parasites, p. 218; it will, therefore, be sufficient to mention here that they possess an outer wall or envelop, formed of condensed areolar tissue and that of the surrounding structure; within which, and rather loosely adhering to it, is the proper membrane. This is white and laminated, and is itself lined internally by a softish layer in which the echinococci are developed. The cavity of the primary cyst is occupied in some instances by a transparent limpid fluid only; in others, and the majority, it also contains a numerous progeny of secondary cysts, which may themselves contain another generation. Dr. Budd mentions the interesting fact, that in cases where suppuration has occurred in the cavity of the primary cyst, the secondary hydatids, though floating in purulent matter themselves, contain a perfectly limpid fluid. He also points out characters whereby to distinguish between an abscess and a suppurated hydatid cyst, in the differences which the cystic membranes in the two cases present. That of an abscess consists of dense fibroid tissue, is not laminated, and never contains calcareous matter. The hydatid membrane does not adhere so firmly to the surrounding tissue, is markedly laminated, and in old cases contains very often plates or grains of calcareous matter in its coats. When an hydatid tumor has evacuated its contents as above described,

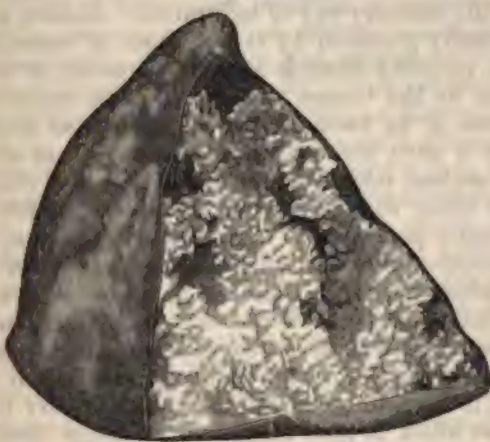
[1] Some doubt may exist whether the purulent-looking fluid contained in the cyst is always true pus. In a case occurring at St. Mary's Hospital, the matter from the interior of a large cyst, which had to the naked eye all the appearance of pus, was found under the microscope to consist of much granular and oily matter, with some cholesterin, and numerous utterly irregular granular masses. There were no true pus-globules.

it may collapse and a cure be effected; but if its walls are very thick and firm, and the cavity large, its obliteration in this way may be impossible, and thence there is too much reason to fear that, owing to the entrance of air or other matters, suppurative inflammation of the sac will be excited, and the drain exhaust the strength of the patient. But an hydatid cyst may come to a spontaneous cure in a different way: its proper membrane, instead of secreting a watery fluid may produce a putty-like matter, consisting of phosphate and some carbonate of lime, with cholesterin and albuminoid matter. This accumulates within the sac, or sometimes around it, imbeds the secondary hydatids, and causes them to shrivel up and perish. Such a change reminds one forcibly of the cretification of tubercle, which is often observed in cases where the tubercular dyscrasia has ceased, and the deposited matter has been partially absorbed. Hydatids in the liver are not unfrequently associated with hydatids in other parts, in the lower lobes of the lungs, or in the spleen, or in the mesentery; in such cases, Dr. Budd is inclined to regard the hepatic cyst as the parent, and the others as originated from germs conveyed from it. The arguments which support this view are, the greater apparent age of the hepatic cyst, and the circumstance that the one "in the liver is associated only with cysts in the lung or in the mesentery;" this seems to indicate rather that the one is derived from the other, than that both are of independent origin. It is also to be remarked that an hydatid cyst often occurs in the liver alone, but rarely, if ever, alone in the spleen or mesentery. These arguments are, certainly, of weight, but seem hardly sufficient to counterbalance the objections, that it is difficult to conceive how a germ from the hepatic cyst should make its way backwards against the stream of blood to the spleen or the mesentery, and that it cannot be considered improbable that a second hydatid should originate in a different locality, in a system which has already shown itself favorable to the production of a primary one.

Cancerous disease is very frequent in the liver: it stands fourth in the list of organs thus affected, according to the Parisian registers; these show that it occurs about once in every sixteen cases of cancer: Rokitansky estimates its occurrence in the liver to be much more frequent; he states "its numerical relation to carcinoma of other organs, as 1 : 5." The above statements do not, of course, refer to primary cancer of the liver only, but include secondary cancer also. Three varieties of cancer have been observed in the liver; colloid is extremely rare, neither Dr. Budd nor Dr. Walsh has met with it; Rokitansky seems only to have seen a single case, and he does not state whether it was primary or secondary. Scirrhus is not very unfrequent, or a transition variety between it and encephaloid: it constitutes roundish tumors, about the size of a large nut, whitish, fibrous, and tolerably firm. Encephaloid is far the most common, and, as in other parts, attains far the largest size. We have seen almost the whole organ converted into a mass of this kind. It sometimes forms separate tumors, sometimes infiltrates the parenchyma. Rokitansky's description of the separate tumors seems to us to apply equally to the scirrhous and encephaloid varieties, as he himself appears to allow. He says: "Their general

form is spherical, though their surface not unfrequently is slightly racemose or lobulated. Those which have been developed in the peripheral portion of the organ, and are, therefore, in contact with the peritoneum, present a flattened or even an indented surface, and the indentation may extend to the very nucleus of the morbid growth. The peritoneal lamina in the indentation is opaque and thickened," probably from having become involved in the cancerous degeneration; it seems to be retracted and drawn in much the same way as the skin is in subcutaneous cancer. The number of the cancerous tumors varies in different cases; they may be solitary or very numerous; primary cancers are usually few, secondary may amount to some hundreds. Dr. Walsh thinks they are most numerous when they occur consecutively to cancer of the

Fig. 283.



Encephaloid growth, occupying a large extent of the liver.

stomach. The scirrhous tumors have scarce any investments of cellular tissue, and adhere closely to the surrounding hepatic parenchyma; the encephaloid have a delicate cyst-like investment, though this does not seem to be constant, and they can be detached more readily. "Infiltrated encephaloid," according to Rokitansky, "always contains obliterated and obsolete bloodvessels, and ducts which are gradually absorbed. The infiltration attacks larger or smaller segments of the viscus; it does not present distinct boundaries, but insensibly passes into the normal parenchyma. It rarely occurs without nodulated cancer." The separate tumors often inclose strata of remaining hepatic structure, a fact which seems to mark a connection between the two forms; some degree of infiltration taking place in each; but in one, the growth simply pushes the parenchyma aside, in the other it spreads its germs everywhere among its elements. The structure of cancerous tumors presents nothing different from that of cancerous tumors in other parts, and is described under the general head of cancer (p. 187). Their degree of vascularity varies: some tumors show very little trace of bloodvessels, others are richly supplied, and are the seat also of interstitial effusions

of blood; to such, the term hæmatoid or fungus hæmatodes is appropriate. Black pigment often is scattered through the substance of the growths, and may be so abundant as to make them appear entirely black. These claim, of course, the appellation melanotic.

Cancerous tumors in most cases produce considerable enlargement of the liver, the atrophy of the proper tissue which they occasion being more than compensated by the amount of their own enlargement; in some rare cases, however, this does not take place, and the liver, though containing many cancerous tumors, is smaller than natural. Masses of cancer which appear on the surface of the liver, sometimes excite adhesive inflammation of the investing serous membrane, and thus become united by false membrane to adjacent parts. Instead of this, they have been known to infect with their tainted fluids the parts in contact with them, and to cause secondary formations of cancer in them, or to extend into them, by the ordinary way of infiltration. Ascites, to some extent, is not unfrequently produced by the presence of cancerous masses in the liver: this probably depends on the obstruction of the portal vein-branches, either by the tumors themselves, or by cancerous matter developing in them, or by fibrinous effusion coagulated within their channel. Jaundice is often observed in cancerous disease of the liver; its production, doubtless, takes place in the same way as that just noticed; the gall-ducts being obstructed, and the escape of bile from various parts of the organ prevented. When the masses are so situated as to press on the common duct leaving the others free, enormous distension of the gall-bladder may take place—it has been seen as large as the fœtal head; such a result, however, is more likely to be produced by cancerous disease of the head of the pancreas, than by growths in the substance of the liver. Primary cancer of the liver is stated by Dr. Budd, seldom, if ever, to occur before the age of 35: from this to 55 is the epoch at which it most frequently manifests itself. Secondary cancers of the liver may occur at any age: they seem, according to Dr. Walsh, to affect a preference for the superficial parts of the organ. They are believed to be produced by the transportation of germs in the blood, or by the medium of the lymphatics; this, doubtless, is often true, but we certainly are of opinion that fluid cancerous blastema is quite as adequate to their production as any solid particle, and this cannot but be absorbed by blood passing through a malignant tumor. There seems evidence to show that when the part primarily affected returns its blood to the liver directly, as in the case of cancer of the stomach, the infectious matter is all detained there, and tumors are not formed in other parts; but when hepatic cancer is consecutive to cancer of the breast, its development must then depend on the absorbed matter finding a suitable nidus in that organ. A sort of spontaneous cure of hepatic carcinoma has been occasionally observed, the morbid growth becoming converted into a fatty mass, doubtless by a change of the nature of fatty degeneration.

ABNORMAL CONDITIONS OF THE BILIARY PASSAGES.

Malformations.—The gall-bladder is sometimes wanting—in animals it has been found double; its shape may be variously deformed; its duct, as well as the common duct, may probably be imperforate. The cystic and hepatic duct may remain separate, and communicate either both with the duodenum, or one with the duodenum and the other with the stomach. The mucous lining of the gall-bladder and ducts is often attacked with inflammation, which may extend from the duodenum, and spread upwards along the ducts. It is often of the catarrhal kind, and is essentially similar to the affection of the gastro-intestinal mucous surface; like it, subsiding after a time, and leaving no traces of its existence behind. The effects it produces will be those of vascular injection, some degree of tumefaction, shedding to a greater or less extent of the epithelium, and casting off of mucous corpuscles and various forms of immature epithelia, together with exudation of *liquor muci*, of various degrees of viscidity and tenacity. The gall-bladder alone may be the seat of acute idiopathic inflammation, or this may be excited by unhealthy bile, or, perhaps, by the irritation of a calculus. The result of such inflammation may be closure of the cystic duct, and conversion of the gall-bladder into an abscess. If the catarrhal inflammation, or that set up in any other way, attain a certain degree of intensity, it causes the effusion of muco-purulent or purulent matter, and at the same time it seems to induce paralysis of the contractile coat of the biliary ducts; these tubes, thus weakened, yield to the distending force within of the accumulating secretion, and become dilated at intervals into cyst-like pouches, filled with muco-pus tinged yellow or green by bile. The dilatation will, of course, be promoted, if the common duct, or the hepatic, is obstructed by a calculus, or in any other way. After such pouches have existed a certain time, they become entirely cut off from the duct in which they originated, the tube becoming obliterated by adhesion, and their contents then undergoing certain changes. Thus, the muco-purulent matter may be converted into a clear glairy fluid, more or less tinged with bile; we have recently observed a case of this kind, and though we were some time in doubt as to the nature of the cyst, which was found in a healthy liver, we were soon convinced by detecting particles of columnar epithelium in the matter lining its surface; besides the fluid in this case, there were several small whitish masses attached to the inner surface, consisting of a semi-homogeneous, semi-granulous, soft substance, containing imperfect celloid forms. It seems probable that, had the person survived longer, these whitish masses would have increased considerably, so much as to fill the cyst, and that in this way one of those peculiar tumors would have been produced which Dr. Budd has called "knotty tumors of the liver," and which he believes to be formed within the ramifications of the hepatic ducts. He describes them as firm, white nodules, surrounded by a distinct cyst, and containing a cheese-like substance, in the centre of which is a small mass of concrete biliary matter; they are evidently situated in portal canals, and have often been mistaken for cancerous tumors. We think it is too

much to assume that all such tumors as contain a glairy fluid have originated in catarrhal inflammation; probably the morbid, cyst-producing action is in many cases of a more chronic kind, and the fluid is glairy from the first. In a boy who died with pneumonia supervening on a tuberculized state of the lungs, we found the liver, with the exception of marginal oily accumulation in the lobules, and a somewhat atrophied condition of the cells, apparently healthy, except that here and there throughout its substance there were seen green-colored spots of the size of a pin's head. These seemed to exist about the termination of the minuter portal canals, and were, doubtless, connected with the terminal ducts; they consisted of yellow and orange or reddish pigment coloring-matter, heaped up together and forming a mass which encroached on the parenchyma; in one of them, columnar epithelial particles were seen, proving that a duct was involved in it. The cells contained no yellow matter, so that it was evident that these green masses had been produced by a morbid action set up in the minute ducts. Ulceration of the gall-bladder is not unfrequent; it may occur as a consequence of suppurative inflammation, or be set up in an organ which has been the seat of chronic disease, or occur in the course of remittent or typhoid fever, or be produced by the irritation of calculi, and probably also by that of unhealthy and acrid bile. The ulcers are sometimes small and numerous, sometimes there is but one large one; they are sometimes attended with sloughing of the coats, and sometimes go on to perforate the wall completely. When this happens, the bile, if the gall-bladder contain any, escapes into the cavity of the peritoneum, and rapidly excites fatal inflammation. If, from long closure of the cystic duct, the gall-bladder contains no bile, but only a mucous or serous fluid, this does not escape so rapidly, and the inflammation is more limited to the neighborhood of the liver. When ulceration is excited by the presence of gall-stones, it usually happens that the bladder becomes adherent to some adjacent part, commonly the colon or duodenum; and, as the process advances, a communication is established between the two viscera, by means of which the calculus escapes into the bowel, and may be discharged. We have, however, seen a case in which fatal obstruction of the intestines was occasioned by a gall-stone, which had probably escaped by a fistulous opening from the gall-bladder. We do not know much of ulceration of the ducts; the smaller branches are so rarely examined, that its existence may have been overlooked. Dr. Budd records one very interesting case, in which an ulcer of the common duct made its way into the superior mesenteric vein, close to its termination in the portal; the result was phlebitis of the vessel, and purulent formations in various parts of the liver and the lungs, as well as in the skin and subcutaneous tissue of the head and face, and in some other parts also. If inflammation, attended, probably, with some amount of ulceration, attack the cystic duct, obliteration of its channel may be the result; or the same may be occasioned by a gall-stone lodging there. The gall-bladder now has become a closed sac; the bile which it contained is gradually absorbed and replaced by a mucous or glairy fluid; this is at first so abundant as to convert the bladder into a dense capsule, resembling, according to Rokitansky, the sound of fishes; but afterwards this fluid is reabsorbed,

and the gall-bladder contracts and shrivels. Cholesterin is often present in great abundance in the fluid contents of such gall-bladders; this is especially the case when the coats are diseased and thickened. The loss of the biliary reservoir seems to have no injurious effect on the health. The duct. com. choled. may be closed by concretions, cancerous growths, or croupy exudation: the outflow of the bile being thus prevented, it collects within the ducts, and causes general dilatation of them. In such cases, Rokitansky says, the liver is in a condition resembling that of yellow atrophy, the parenchyma of a dark-yellow or green color, turgid, though pulpy and friable. Dr. Budd, in the case he records, describes it as of a deep olive, finely mottled with yellow; the tissue flabby, but not easily broken down; the lobules undistinguishable. The cells in this and in another case were destroyed, and only granular and oily debris, mingled, in Dr. Budd's case, with yellow matter, remained. In two cases which we have observed, the cells were not destroyed; they had a yellow or greenish tint, and were rather stunted, but not at all broken up. We injected in one a large dilated duct, and obtained the important result *that the terminal ducts were not dilated, and were of about the same size as in healthy livers.* Rokitansky says that "this affection invariably proves fatal with symptoms of biliary infection of the blood and consequent cerebral disease, which is often combined with exudation on the arachnoid, with intense icterus, and extreme pain in the liver." In Dr. Budd's case, there were no symptoms of cerebral poisoning, and the mind remained clear to the last.

Croupy inflammation occasionally, but very rarely, attacks the gall-ducts. "It gives rise to tubular exudations, in which the bile forms branched concretions, which block up the passages, and thus cause dilatation of the capillary gall-ducts." The coats of the gall-bladder may become œdematous in dropsy, or the subserous tissue infiltrated, as in peritonitis; increased deposit of fat may also take place in the latter situation, and, perhaps, induce (coincide with?) fatty degeneration of the muscular layer. Rokitansky also notices the formation of osseous plates in the thickened parietes of gall-bladders which have been the seat of inflammation, an and increased production of fibroid tissue, which may be so firm and white as to give a cartilaginoid appearance. The biliary ducts very rarely contain tubercle. Cancer sometimes extends to the gall-bladder from the liver; or, it is said, may occur in it primarily; it forms nodules in the submucous tissue, or infiltrates the mucous membrane; more commonly, its wall is perforated by growths in the liver, which push their way into its cavity.

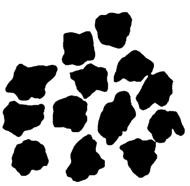
ABNORMAL STATES OF THE BILE.

A brief account of the various unhealthy states of the biliary secretion will properly follow here. It must be premised, however, that our knowledge of these states is extremely imperfect, from the bile being, unlike the urine, almost inaccessible in the living subject to our observation, and from the changes which it spontaneously and rapidly undergoes. It may be secreted in too great quantity, as commonly

happens to Europeans on first arriving in India, and occasions a bilious diarrhœa. The same thing often happens in our own country in autumn, and from the same cause, probably, viz: increased excitement of the liver. The converse of this sometimes occurs, bile is secreted in too small quantities, or for a time ceases to flow altogether. This produces pain and uneasiness in the bowels, which are relieved when the bile flow returns. It seems that bile is the natural and healthy stimulus to the intestines, and that, if it is absent, the other contents, acid, and, perhaps, in other ways irritating, tease and distress the sensitive mucous membrane. We cannot judge correctly of the bile that has been poured out of the liver by that which we find in the gall-bladder, for the latter may be unnaturally dark and viscid, or otherwise altered, simply in consequence of its continued sojourn there. Still, in many cases, we have no other source of information, and from this we must form the best judgment we can. In cirrhosis, Dr. Budd says, the bile is often thin or serous, and of an apricot or orange color; in other similar cases it has its natural appearance. Sometimes it is black and thick. In yellow atrophy, the gall-bladder generally contains but little bile, often only some mucous fluid, tinged yellow or green: the hepatic ducts have been found quite devoid of any biliary tinge. In cases of fatty liver the bile is sometimes unusually pale. We have seen it, however, of a deep greenish color; and this is also the case in fishes in which the fatty condition is natural. In the lardaceous or bacony condition, Lehmann speaks of the bile as light-colored and watery. In tuberculosis, the bile is often found poorer in solid contents, but sometimes also more rich in them. Extensive inflammations, especially pneumonia and diabetes, are said to render the bile more watery. This occurs also in some cases of typhus and disease attended with dropsy. The solid contents, according to Lehmann, of the bile are increased in diseases of the heart, and those abdominal (others also?) affections in which the motion of the blood in the larger veins is delayed. The bile in malignant cholera is very thick and tenacious, and is like the blood manifestly drained of its water. In this disease, and in Morbus Brightii, urea has also been found in the bile. Albumen seems to have been found occasionally in bile; Lehmann says that it has been observed in cases of fatty liver, of Bright's disease, and in the embryonic state. The quantity of mucus is often relatively increased when the bile is very dilute; this has been noticed in typhus fever. Free oil is occasionally seen in bile, and probably always might be during decomposition of this fluid. The bile is very rarely acid; this has been observed in cancer and typhus; it probably depends on some of the acids being detached from their bases. The foregoing statements are very unsatisfactory; and, perhaps, the only definite conclusion that can be drawn from them is, that there is no constant relation between the condition of the parenchyma and that of the secreted bile, nor between the latter and the greater number of the various diseases to which the body is liable. Obviously, they tell us nothing as to the varying condition of the resinoid biliary acids, the essential constituents of the bile. Two conditions, however, of the bile in the gall-bladder deserve our close attention, on account of the important consequences which often result

from them. One is, the bile being so loaded with coloring matter from concentration, or other causes, that a deposit of this substance takes place; the other is, the bile containing a large quantity of cholesterin. From these two substances all biliary calculi almost are formed. They are far most common in the gall-bladder, but also occur in the ducts, both within and without the liver. "Their[1] form and surface vary much. Single calculi are commonly round, oval, or cylindrical; when very large so as to occupy" the entire cavity of the gall-bladder, they are frequently slightly curved; "if many exist together they mutually prevent their enlargement, and, in consequence of the friction and pressure they exert upon one another, they assume cubical, tetrahedric, prismatic, or irregularly polyhedric shapes, with convex or concave surfaces. The calculi found in the ducts are generally cylindrical, occasionally branched, or entirely amorphous. The texture of the calculi may be uniform or varied, in proportion as they consist of one substance or of several layers. Many show no distinct arrangement; some have an earthy pulverulent fracture, or a fibrous, striated, laminated, micaceous texture, as is particularly observed in calculi consisting of cholesterin." Gall-stones are not of any great degree of consistence; they may sometimes be compressed easily between the fingers: they are rather light, but not so much so as to float in water. Their color varies from a milky-white to various shades of green, yellow, or brown: internally, they often present an alternation of different-colored laminæ. They may be said to consist generally of cholesterin, mingled with a combination of pigment and lime in various proportions. Large gall-stones, with the exception of their nuclei, consist almost entirely of cholesterin, and are, therefore, whitish and crystalline; their sectional surface presenting a number of striæ radiating from the centre. Small gall-stones, resembling grains of black pepper, of an irregular, tuberculated form, and almost black color, are occasionally found: they consist almost entirely of pigment and earthy matter, the carbonate and phosphate of lime. Cholesterin generally forms the principal mass of biliary calculi; it often alternates with layers of pigment, and almost always itself surrounds a nucleus of the same matter.

Fig. 284.



Small, irregular gall-stones, composed of inspissated and altered bile cemented by mucus.—From Dr. Budd's work.

The secretion of a large quantity of cholesterin does not seem to be the essential and adequate circumstance for the formation of gall-stones; the gall-bladder sometimes contains mucus loaded with sparkling tablets of this substance, without any trace of calculi. It seems most probable that pigment-granules cemented together by mucus first constitute a nucleus, round which cholesterin afterwards is deposited in layers. Other matters, however, may serve as a nucleus; blood, a portion of a distoma, or a lumbricus, or even a pin, are said to have been found in this situation.

[1] Rokitansky, vol. ii. p. 162.

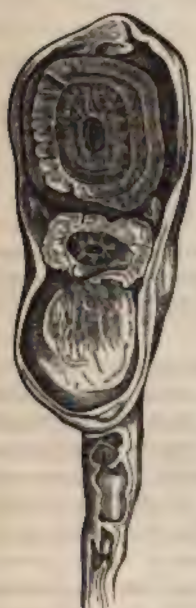
Fig. 235.

From a gall-bladder, which was shrunken, a calculus being impacted in the cystic duct.

(a) Cholesterin tablets.

(b) Glomeruli.

Fig. 236.



Gall-bladder and cystic duct, containing calculi, which have a crust of pure cholesterin. The two upper are divided. — From Dr. Budd's work.

Gall-stones are not peculiar to, or especially associated with, any condition of the liver—they are said to be most frequent with cancer, but very often occur in other states; they are more often found in females than males, in the proportion of 4 or 5 : 1—rarely before, but often after the middle period of life. A sedentary life, and obese condition of body, are favorable to the formation of gall-stones; they are not, however, unfrequently present in lean and temperate persons. As cholesterin is a variety of fatty matter, this might seem surprising, did we not remember that its formation may be referred to a kind of fatty degeneration, as well as to the presence of an increased quantity of oil in the system. Cholesterin is certainly secreted often in large quantity by the thickened coats of the gall-bladder, and by additions from this source the large solitary calculi are probably formed; there is no doubt, however, that in other cases it is deposited from the bile, owing to the decomposition of the taurocholic acid or its salts, by which it is naturally held in solution. Calculi are often loose and free in the cavity of the gall-bladder; sometimes they are attached to its surface by exudation, or included in compartments formed by organized lymph. Small ones are sometimes, also, contained in saccular dilatations of the mucous membrane, and may appear to lie external to the cavity of the gall-bladder. The following effects are produced by biliary calculi: They become impacted in the cystic duct, and occasion its obliteration, the gall-bladder undergoing the changes that have been described. The same thing occurs, but much more rarely, in the common duct, which is straighter and wider; great distension of the gall-bladder and ducts, and occasionally rupture of the former, then take place. If, on account of the angular shape of the stone, the duct is only partially obstructed, the same effects are produced, but in a less degree. While lodged in the gall-bladder, calculi may excite irritation, thickening, inflammation, and suppuration of its coats, and sometimes ulceration. Of these we have lately spoken.

Acephalocysts, and the distoma hepaticum, occur in the gall-bladder. For a description of them, *vide* art. *Parasites*, p. 199.

CHAPTER XXXIV.

ABNORMAL CONDITIONS OF THE PANCREAS, AND THE OTHER SALIVARY GLANDS.

These are not very numerous. Congenital deficiency is observed only in very imperfect monstrosities, and excess of development is very rare. *Hypertrophic* enlargement, Rokitansky states, is altogether unusual, and when it does occur, affects chiefly the cellular tissue, which is interwoven with the glandular tissue. We have, however, examined one specimen in which the ultimate vesicles were stuffed with epithelium to such a degree, that their investing fibroid envelops appeared stretched and distended, and the whole gland was of a very remarkable density. We think minor degrees of this condition are not uncommon.

Atrophy of the pancreas takes place in some instances spontaneously, chiefly in advanced age; or it may result from chronic inflammation, or fatty degeneration. The organ may be soft and lax, or of leathery consistence.

Inflammation, at least in the acute form, but rarely attacks the pancreas; it is, however, not infrequent in the other salivary glands, where it constitutes the disease termed mumps. We have seen inflammation and suppuration of the parotid gland occur as the result of fever. The phenomena of inflammation are the same here as in other similar parts. The gland swells considerably, partly from the congestion of its vessels with blood, partly from exudation into the areolar tissue which envelops it. In the ordinary case of mumps, suppuration rarely takes place, and simple resolution occurs; but when the result is less favorable, the glandular structure becomes, in a measure, fused with the interstitial exudation, and probably penetrated by it also, and the whole mass softens and breaks down into purulent matter. This had occurred in the case of fever above alluded to. The suppuration may affect the whole gland, or be limited to distinct spots, and form an abscess. "Chronic inflammation induces condensation, induration of the cellular tissue, obliteration of the acini, and either permanent enlargement, or subsequent atrophy of the gland." A specimen of this kind which we examined, was in the following condition: There was a very large quantity of coarse fibrous tissue surrounding and enveloping the lobes and lobules of the gland. In this there was much irregular fatty deposit, appearing more like masses of concrete, fatty matter, than true adipose tissue. The gland-tissue was more or less atrophied, not nearly so apparent as natural, the ultimate vesicles were not well seen. The epithelium looked coarse-grained, and contained much oil. The fatty deposit seemed to have

taken place in a secondary manner as the result of the shrinking of the gland.

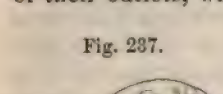
Fatty degeneration of the pancreas is described by Rokitansky as frequent in drunkards, associated with fatty liver. It is not, however, a degeneration of the same kind, but rather, from this account, seems to take place by intrusion of the surrounding adipose tissue on the wasting organ. Serous cysts occur occasionally in the pancreas, and the other salivary glands.

Cancer does not select these glands as one of its ordinary sites. It does, however, affect them not unfrequently both primarily and secondarily. Scirrhus and encephaloid are the only two forms which occur. It is probable that, in several of the cases reported as cancer of the parotid, the disease was really seated in some of the adjacent or imbedded absorbent glands. The head of the pancreas, where it is embraced by the duodenum, appears to be the part of the organ most frequently affected. As a result of the growth of the tumor, the ductus choled. sometimes is obstructed, and jaundice is produced. The disease may extend much further than this, according to Dr. Walshe, implicating "the duodenum, the omentum, mesentery, liver, and even the supra-renal capsules and kidneys." Rokitansky says, "that the secondary affections of the salivary glands, by an extension of the disease from adjoining organs, and in the case of the pancreas especially, by an extension from the scirrhous pylorus, is very common.

Salivary fistulæ are usually caused by the progress of ulceration. Thus, a perforating ulcer of the stomach may make its way into the pancreatic duct, and the same with regard to the duct of Steno, which is oftenest perforated.

Dilatations of the ducts are produced in consequence of obstruction of their outlets, while the secretion accumulates and distends the canal. The obstruction may depend on an external tumor, or a mucous plug, or on a calcareous concretion. Sometimes the dilatation occurs at several separate points; sometimes it forms fusiform "or closely-set expansions, partially separated from one another by valvular folds formed by the coats of the duct." The salivary concretions, or calculi, are described by Rokitansky as "white, friable, and either round, oblong, cylindrical, or obovoid; in size varying from that of a millet-seed or pea, to even that of a hazel-nut. They are either solitary, or, if small, frequently very numerous (twenty and more); and they are composed of phosphate and carbonate of lime, held together by animal matter." The saliva from which they are formed by deposition, must be, as Dr. Walshe remarks, in an unhealthy state; for while the concretions consist chiefly of phosphate of lime, sometimes containing 94 per cent., there exists very little of this salt in the normal secretion. "It becomes, therefore, extremely probable that the excess of phosphate is generated through the influence of irritation of mucous membrane."

Fig. 237.



Salivary calculus of considerable size; removed by operation.

ABNORMAL CONDITIONS OF THE DUCTLESS GLANDS.

Of the Spleen.—This organ is generally absent in acephalous monsters; sometimes it is wanting, together with the stomach, or the fundus of the stomach, in subjects otherwise well developed; or it may be itself alone in a rudimentary state. Small supernumerary spleens, which are often met with in the vicinity of the organ, are not to be regarded as instances of its multiplication, but of its subdivision. The spleen is liable to very great variations in size, probably more than any other organ of the body. This depends chiefly on the very large size of its vascular system, and on the great quantity of yellow, elastic fibre contained in its structure, which allows it to be distended to a prodigious extent. Most, if not all, hypertrophies of the spleen, however, are produced not only by engorgement of the vessels, but by an alteration and increase of the red, pulpy parenchyma which they traverse. This parenchyma consists of nuclei, with granulous matter in small quantity, and some slight traces of cell-development. It evidently is not a very highly organized substance, like a muscular fibre, and will easily admit of increase or diminution. We shall here only mention the degrees of change in size which the spleen may undergo. Rokitansky states that "the spleen not unfrequently measures sixteen inches in its long, seven inches in its short diameter, and four inches in thickness; its weight may amount to 13½ lbs.," or even, as others affirm, to 20 lbs. and upwards. The opposite change of atrophy may reduce the spleen to the size of a hen's egg, or a walnut. The *form* of the spleen is rather various. It may be tongue or platter-shaped, or cylindrical or globular. One of the most important circumstances to note under this head, is the great frequency of notches in its anterior border, which may be felt through the abdominal parietes when the organ is enlarged. The spleen is liable to various *displacements*, some of which are congenital, others the result of disease. Haller found it lying by the side of the bladder in a child one year old, Desault in the right side of the thorax in a new-born infant. It has been found in the left thoracic cavity when the diaphragm was absent, and external to the abdomen in large umbilical herniæ, or where the abdominal parietes were fissured. Displacement may ensue from the enlargement or distension of adjacent parts, or from its own increase in size. Sometimes in the latter case it descends to, and slides off, the ilium, "so as to occupy a diagonal position in the hypogastrium, and extend over the right ilium."

Wounds and Ruptures of the spleen occasionally happen from injuries or accidents. The only point of interest respecting these is, that there appears, from the observations of Mr. Athol Johnson, ground to believe that, under careful management, they do not necessarily prove fatal. Spontaneous ruptures of the spleen have also occurred in conditions of intense congestion, and when the texture of the organ was probably weakened; as in typhus, cholera, and the cold stage of ague. These always prove fatal.

With regard to the textural changes in the spleen, Rokitansky remarks "that they almost always arise from certain anomalies of the

blood, which, though little known, bear a remarkable and positive relation to the spleen. The spleen may, in fact, be considered as the most sensitive test for a variety of dyscrasic states of the fluids." In this we doubt not that there is much truth, and think the view that a certain anomalous condition of blood is necessary for the production of great enlargement of the spleen is much supported by the circumstance that the organ is scarce ever materially enlarged in cases of cirrhosis of the liver, when the current through the splenic vein must be so greatly impeded.

Hyperæmia of the spleen occurs both from mechanical causes, and from that just noticed. Rokitansky remarks that, though it occurs in organic diseases of the heart and in hepatic obstructions, it does not amount to the extent, nor take place so frequently as might be expected, and he accounts for this by the deranged circulating fluid having no affinity for the tissue of the spleen. In the bodies of drowned persons the spleen is found gorged and distended with blood. This blood, no doubt, might all be washed out by injecting the vessels with water, and the organ would return to its normal size; but when congestion comes to be permanent, the exudation which takes place in the red parenchyma becomes organized into similar celloid substance, and the spleen is then truly hypertrophied.

An *anæmic* state of the spleen is observed in the highest degree when the parenchyma is infiltrated with bacony matter, to be presently noticed; but it also exists in many atrophied conditions, which very often depend on, or coincide with, a development of the nuclei into fibres.

"Primary *inflammation* of the spleen," Rokitansky says, "is as rare as spontaneous primary phlebitis; secondary, as frequent as secondary phlebitis." Primary inflammation of the spleen, unless ending in resolution, gives rise to an exudation of laudable pus or fibrin. The pus may be contained in a circumscribed abscess, and thence become obsolete, or the cavity may go on enlarging until the abscess makes its way into the left thoracic cavity, the stomach, the transverse colon, or the peritoneum. When the latter event happens, circumscribed peritonitis often forms a sac for the pus with the aid of the surrounding parts. Secondary splenitis seems to be identical with pyæmic deposits, and is stated by Rokitansky to consist in nothing more than the (purulent) "metamorphosis of an infected coagulum within the channels of a vascular ganglion." The deposits are well defined, always situated at the periphery, usually of a cuneiform shape, the apex directed inwards; their color is considerably darker than the surrounding tissue, and their consistence firmer. A ring of reactive inflammation is often set up around them. The process may terminate either in the case of a benignant fibrinous exudation in conversion of this into a cellulo-fibrous callus, which contracts and causes a cicatrix on the surface; or in the case of a less healthy exudation in the conversion of this "into a puriform, creamy mass, or into a sanious, greenish, greenish-brown, or chocolate-colored pulp." Rokitansky notices the frequent occurrence of the above affection in "inflammation of the internal vascular coat, and particularly" in endocarditis. As he makes no special mention elsewhere of the fibrinous block so common in the splenic parenchyma, we

conclude that he comprises it among the phenomena of secondary splenitis; but we cannot help doubting whether this is correct. To us it seems much more probably to be a simple exudation of fibrin, which takes place in consequence of the blood being surcharged with this product; and we should regard the simultaneous deposition of fibrin on the endocardium of the valves of the heart as a mere coincidence, and not in any degree as a cause. M. Simon is inclined to consider that, in many cases, disease of the artery leading to the part is the cause of the deposit. The appearance of these deposits is that of a circumscribed yellowish mass, with a surrounding margin of darker or lighter red congestion, of increased consistence, so as to be readily detected on handling the part, and exhibiting under the microscope a confused mass of granular with more or less oily matter, infiltrated among the remains of the parenchyma. They very commonly undergo fatty degeneration, and this appears to be the way in which they are removed.

Among the various *enlargements* of the spleen more or less connected with hyperæmia, that occurring in typhus deserves to be noticed. The parenchyma, in very marked cases, is exceedingly soft, almost breaks up under the hands; its color is a dirty red, varying from different shades of depth to a light chocolate; its size is greatly increased; but it shows, under the microscope, no very noticeable alteration of its structural elements. There may be some increase in the quantity of diffused granulous matters; but the nuclei appear quite natural. The change is one better judged of on a large than on a small scale. We have examined, at different times, numerous specimens of greatly enlarged spleen, but we have found little that could be regarded as characteristic of the several alterations. This is not surprising; it could not be expected that specific differences in the blood should mark themselves by corresponding varieties of form in the cell-growths of their exudations, any more than that the syphilitic virus should be detected by some special modification of the pus of a chancre.

In a case of Leucocythemia, recorded by Dr. Chambers in the Report of the Pathological Society for 1846–47, it is stated that the spleen, which measured 14 inches by 4 or 5, was extremely dense, "exhibiting on a section a beautiful mottled appearance; but under the microscope presenting no obvious deviation from the normal character. In a similar case we noted that the nuclei of the parenchyma were more granular than usual, and were often in process of cell-development, but the same change often occurs in other diseases. In a man dying with ulcerations of the colon, producing excessive diarrhœa, and who had formerly passed large quantities of lithic acid, the greatly enlarged spleen presented a perfectly uniform smooth surface, of a rather light red color, and exhibited under the microscope no peculiarity of structure, except that, together with the normal elements, there were mingled numerous largish crystals, probably those of triple phosphate. In a female dying with cancerous growths in the fauces and in the lungs, the spleen was so enlarged as to weigh four pounds twelve ounces. Its cut surface exhibited an irregularly raised appearance, being formed of elevations of a dull whitish color, with intervening red streaks. The whitish elevations consisted of pale, colorless, nuclear corpuscles, closely resembling the

normal corpuscles of the Malpighian bodies. The red parenchyma had almost disappeared, there were only a few vessels seen running between the hypertrophied Malpighian masses. There was a considerable amount of black pigment deposited in the course of the vessels, and some crystals, like those seen in the former case, were also present. The examination of these two specimens seem to warrant the conclusion that, in the former, the red parenchyma was the seat of the hypertrophy, the Malpighian corpuscles in the latter. The two following instances seem to be examples of the infiltration of the splenic parenchyma with fibrinous matter, rather than of a true hypertrophy of it like the preceding. It is especially to be observed that they were general, and not confined to one part of the viscus. Dr. Ogle has recorded, in the Report of the Pathological Society for 1851–52, the case of a man who died with pleurisy in connection with granular degeneration of the kidneys. The spleen weighed seventeen ounces, and was of firm, solid texture throughout. "In its substance were three or four large patches of a yellowish-colored deposit, having a slightly pink hue towards their central parts. These existed at the peripheral portions of the viscus, and penetrated to some extent into the interior. Moreover, the whole viscus was of a mottled color, owing to an extensive infiltration of a lightish-colored material, which gave to it its firm, unyielding character. Examined by the microscope, the deposits proved to consist of amorphous and granular matter, larger fatty granules, with great numbers of small, oval, and elongated cells, having a nuclear appearance, along with an admixture of what seemed to be the natural cells of the organ. No indications of ulterior development or organization existed in the deposit. The investing fibrous capsule of the spleen was thickened and rendered opaque, having several tough bands of false membrane attaching it to contiguous parts;" and the lining of the splenic artery contained atheromic deposit. In the following case, a similar deposit had taken place both in the liver and spleen, giving to both organs a great increase of firmness and toughness. The cells of the liver were almost entirely destroyed, and replaced by a fibroid tissue containing in its substance numerous nuclear and celloid particles. The spleen weighed three pounds six ounces, and measured fifteen inches by ten and a half; it was very bloodless, but its arteries appeared healthy, though the aorta was highly atheromatous. The cut surface of the organ had a peculiar gray, uniform aspect, with some small red specks and dots scattered over it. Under the microscope it showed a granular, homogeneous basis, having a slight tendency to fibrillate, with numerous nuclear corpuscles and much oily matter dispersed through it. The nuclear corpuscles resembled exactly those of the healthy organ. In a third form of chronic enlargement of the spleen the character of the alteration is distinctly marked by the presence of a peculiar form of deposit which is described by the terms lardaceous and bacony. It often exists at the same time in the liver and kidneys, and always appears in connection with a lowered condition of the organic power. It appears, under the microscope, as irregular fragmentary masses, homogeneous, translucent, and sub-refracting, mingled commonly with more or less of the structural elements of the part in which it is deposited. It has appeared to us sometimes to be deposited in the situa-

tion of the Corpora Malpig., so that there are seen spots or circular spaces which are separated from each other by the remains of the parenchyma. The organ is often much enlarged, is remarkably bloodless when extensively invaded by the deposit, and at the same time is brittle, and gives a peculiar resinoid fracture. We may recapitulate the foregoing account of chronic enlargements of the spleen, by enumerating them as resulting from (a) true hypertrophy, (b) infiltration with fibrinous deposit, (c) a condition compounded of hypertrophy and infiltration, and (d) deposition of lardaceous matter. Rokitansky states that many cases of splenic enlargement "depend upon the formation of certain corpuscles, in addition to the existing hyperæmia." He denies that these corpuscles are identical with the Malpighian, but his own description of them shows that they are very similar; and as we know, from our own examinations, that these do occasionally exist, we are much inclined to believe that those to which Rokitansky refers are no other.

The fibrous capsule of the spleen is very frequently the seat of *chronic fibroid thickening*, which sometimes proceeds to a very great extent. The thickened membrane has much the aspect of cartilage, but none of its real characters. Usually, the thickening extends pretty uniformly over the surface, leaving, however, here and there, spots less affected; sometimes it forms nodular masses grouped together. In one case we examined carefully, it was very apparent that the thickening had taken place on the inner surface of the capsule, at the expense of the parenchyma, and we are inclined to think this is generally the case. Certainly, the process is distinct from the formation of bands of adhesion to adjacent parts. Fibroid tumors of the parenchyma are rare. Ossification of the thickened fibroid layers takes place rarely, except in very old persons. Fibrinous deposits in the parenchyma may, together with other alterations, cretify. Phlebolithes may form in the venous channels.

Fig. 238.

Fibroid thickening of capsule, encroaching on the dark parenchyma. Vertical section.

Tuberculous matter is, for the most part, deposited in the spleen only in acute universal tuberculosis; it occurs more frequently in children than in adults, in the proportion of 40 : 13. It appears both in the form of gray granulations, miliary crude tubercles, or yellowish cheesy masses, of the size of a pea and above. In acute tuberculosis, the spleen is described by Rokitansky as becoming swollen and softened, much as in the typhous state. The organ was much enlarged in the last case we witnessed, but it was distinctly observable that there was no congestion around the tubercles. Their substance consisted of an amorpho-granulous matter imbedding oily molecules and freely-formed nuclei. Rokitansky notices the formation of a pseudo-cyst round tubercles, and the not unfrequent presence of a small central cavity in their interior.

We have occasionally observed small *cysts* in the spleen, or, perhaps, to speak more accurately, in its capsule; in a female of mid age dying with fever, there were several small, firm, nodular prominences on the anterior border; they were of conical shape and lightish red color, appearing like so many growths on the surface; under the microscope, they

Fig. 239.

Masses of crude tubercle in spleen.

were found to be small cyst-like cavities of varying size (one measured $\frac{1}{8}$ inch diameter), oval or spherical, containing numerous large granule-cells floating in a transparent liquid.

Fig. 240.

Cyst in the capsule of the spleen, containing a clear fluid and glomeruli.

Hydatid cysts are sometimes found in the spleen alone, or concurrently "with one in the liver; it rarely attains the size it reaches in the latter organ." Dr. Coley exhibited to the Pathological Society a specimen of the magnitude of a cocoa-nut.

Cancer is rare in the spleen, the only form which it assumes is encephaloid, and this is very rarely, if ever, solitary; it occurs associated with similar disease of the liver, stomach, and omentum."

OF THE THYROID GLAND.

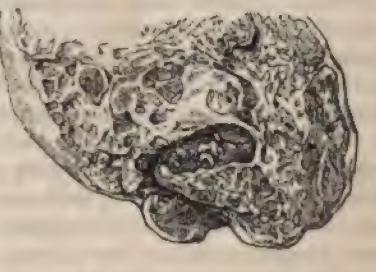
We are not acquainted with any instance of excessive congenital development of this gland, unless the rare occurrence of simple hypertrophy should be regarded as such. Mr. Curling has recorded in the *Medico-Chirurgical Transactions*, two instances of its absence in idiots. The most complete account of its morbid alterations is that given by Professor

Hasse, from which we shall extract the greater part of the following summary: "*Inflammation* of the thyroid is rare. It may attack the organ, either when healthy or when enlarged by previous disease. Its course is more frequently chronic than acute. Within a very brief interval the gland often swells considerably—becomes very bloodshot, tense, and painful—its texture softened and friable, assuming first a brown-red, and ultimately a dingy-gray color." When suppuration occurs, there may be several foci, or one large one involving the whole gland. The abscess may open externally, or into the œsophagus, or into the trachea. After this has occurred, the gland on the side affected shrivels "into a hard, cellular, filamentous knot, which adheres firmly to the skin and the surrounding parts. Sometimes the shrivelling of the one gradually brings on wasting of the other lobe." Andral states that acute tumefaction of the thyroid may come on after violent exertion. *Simple enlargement* of the thyroid is frequent, says Hasse, and for the most part inconsiderable; but it implicates the entire gland, and thus may cause greater disturbance than a more extensive tumor of another kind. "Both lobes of the gland, and even the middle one, swell so as to encroach equally on each side, against the trachea and vessels of the neck." The affection is almost wholly confined to youth, and is frequent about the age of puberty in both sexes—more so, however, in the female, in whom enlargement is especially apt to prevail at the approach of the menstrual period. The essence of simple hypertrophy

Fig. 241.

Bronchocele, from the King's College collection. The œsophagus is seen to be pushed to the right side by the tumor.

Fig. 242.



Section of a bronchocele, showing calcareous deposits.—From the Middlesex Hospital Museum.

seems to be the derangement of the equilibrium naturally existing between the processes of secretion and absorption, that continually go on in the gland. When the former predominates, the vesicles, and of course the whole organ, become distended. M. Coindet relates that a regiment of young recruits were almost all attacked by considerable enlargement of the thyroid, shortly after their arrival at Geneva. On changing their

habitation, and the water which they used for drink, they all quickly recovered. In this instance, and in a multitude of similar ones, it seems beyond doubt that some constituent of the water being absorbed into the blood, acted as a stimulus to excessive secretion into the thyroideal cavities. The effect produced would be quite analogous to that of a diuretic salt. In the chronic and permanent enlargements of the thyroid, a more considerable amount of change takes place. The vesicles of the gland are not only distended by excess of their natural secretion, but, besides being greatly enlarged, are filled with calcareous, atheromatous, and other matters. It is probable that some new vesicles are formed, as well as old ones enlarged. Hasse makes two separate varieties of melicerous degeneration and cystic formation; but we think they run so much into one another that they may be classed together. In the pure melicerous degeneration, the secretion which distends the vesicles, and some of them much more than others, is a tenacious, viscous, jelly-like substance, of the color of honey. The vessels seem to be usually compressed, so that the gland appears bloodless. The change is sometimes limited to certain portions, an interesting instance of the power which the vitality of each individual elementary part has in determining the character of its actions. In the more common cases of enlarged thyroid, the vesicles contain, besides colloid matter, more or less inspissated or fluid, varying quantities of calcareous, often ossiform substance. We have seen crystals approaching the octohedral form in the contents of the cavities, as well as tablets of cholesterin; and fatty matter seems to be not unfrequently present, probably as the result of fatty degeneration. An instance related by Dr. Häen, shows the great variety of appearance which the contents of the cysts may present. He says that, in a frightfully enlarged thyroid, he found almost every variety of tumor existing together. "Here was a steatoma, there an atheroma; in another place a purulent tumor; in another, an hydatid; in one, there was coagulated, in another, fluid blood; on this side was a loculus full of glutinous matter; on that, was one filled with calcareous matter, mingled with tallow." One of the cystic cavities sometimes enlarges prodigiously at the expense of the others; Andral relates having found a thyroid transformed into a cyst with bony walls, filled with a honey-like matter. Another kind of enlargement of the thyroid, consists in the *dilatation of its vessels*, constituting what is called vascular or aneurismatic bronchocele. The veins in particular, writes Hasse, form very dense, capacious, often knotted plexuses, and the whole texture consists, apparently, of a dense coil of vessels. The substance of the gland has almost entirely lost its granular character—it is flabby and dark-red. After death, the tumor collapses considerably. The walls of the arteries and veins are attenuated; the dilated membranes of the vessels contain considerable clots, and capacious cavities are found, filled with black coagulated blood. In a remarkable specimen, which seemed in part to belong to the above class, we found the greatly dilated vessels completely coated with oily matter, so as in some parts to appear like white cylinders by direct light. At the same time, the glandular vesicles were destroyed, and only some traces of their epithelium could be discovered. This, which would certainly be the most important feature of the alteration, is not directly

stated by Hasse to take place in the above description, though it seems inferable from the statement, that the granular (vesicular) character of the gland is almost entirely lost. We have observed in a second case, the same destruction of the glandular structure, and dilatation and oily incrustation of the vessels, but there was a more considerable deposit of earthy matter. *Tuberculous deposit* is rarely if ever found in the thyroid. *Cancerous* disease is also very rare, but has been observed both in the forms of scirrhus and encephaloid. "When primary, the disease is usually of the infiltrated form; when secondary, of the tuberous." Encephaloid growths sometimes attain such a degree of vascularity, that they resemble fungus hæmatodes.

Of the thymus.—Absence of the thymus has only been observed in cases of acephalism. Inflammation is an exceedingly rare occurrence; but Hasse refers to two cases, in one of which an abscess is said to have opened into the trachea. In cases of tuberculosis the thymus is occasionally involved; it becomes considerably enlarged, "firmly united with surrounding parts, and either converted by tubercular infiltration into a hardened mass, or else partially destroyed by softening." Mr. Simon mentions a case in which suffocation was occasioned by the pressure of a tumor apparently of sarcomato-cystic character, in the situation of the thymus; and Sir Astley Cooper met with a case of encephaloid growth in this part. The thymus is not unfrequently found *greatly enlarged;* but the nature of the hypertrophy, as it is called, has not been exactly determined by microscopic observation. It is tolerably certain that this enlargement is not, as has been supposed, the cause of attacks of sudden and sometimes fatal dyspnœa occurring in children. For it has been shown that "thymic asthma," as it is termed may occur with an unnaturally *small* thymus; and that the gland may be greatly increased in size without producing any symptoms of dyspnœa.

Of the supra-renal capsules.—We take the following brief account in part from Rokitansky. The supra-renal capsules may be deficient, especially where there is a deficiency in other organs also. Their absence, however, does not coincide with that of any other organ in particular, and they may be present when one kidney is absent, so that their name must not be taken to imply any correlation of function. Accessory supra-renal capsules are of frequent occurrence. They are sometimes found *hypertrophied*, but the nature of the enlargement does not appear. Their normal condition is one of at least relative *atrophy*, that is, they do not grow and increase in size together with the other parts of the body. Their most usual condition, according to numerous examinations that we have made, is the following: On making a transverse section, there is seen a cortical layer of a whity-gray or yellowish color, often markedly striated, and consisting of cells, more or less laden with oily matter, and arranged in very perfect rows. Within the cortical, and usually not occupying more than one-third of the whole diameter, is the medullary, traversed by the central vein or its branches. This is of a pale opaline, often dark-red color, and consists of large nuclei set in a granulous basis, containing very little oil. There is often much yellowish pigment in the cells of the cortex where it adjoins the medullary portion; these cells resemble very much those of the liver when laden

with yellow matter. The principal indications of atrophy are manifested, we think, by the shrinking and diminution of the medullary substance, and the breaking up of the cortical cells into oily masses. It is interesting to compare the condition of the degenerating capsule with that of an hepatic lobule. In one instance, where a careful search was made, at our request, for the supra-renal capsules in the body of a child six years old, who had died from a burn, no trace of them could be found; but a small quantity of lax, dirty-looking, reddish, infiltrated, areolar tissue, which presented under the microscope only a mixture of altered granulous nuclei, large granulous or oil-holding cells, and a very large quantity of diffused granulous matter imbedding some oil-drops. This was a case of unusually early atrophy. Another indication of atrophy is afforded by the formation of a central cavity, which exists in some cases quite distinct from that of the vein. It is formed, we believe, by the wasting of the medullary substance, and the production thereby of a space which is traversed by bloodvessels, and occupied only by serum, and a little stromal fibre. In this way a lax spongy tissue is first formed, which often is stained of a dark maroon or bistre color, by exuding and altered hæmatin. This tissue is afterwards absorbed, and a cavity with more or less smooth walls is thus produced. Rokitansky says: "Hemorrhage not unfrequently occurs in them, on account of the vascularity of their medullary substance. The capsule is found distended in proportion to the amount of extravasation caused by the rupture of a vein; and according to the period which has elapsed since the occurrence of hemorrhage, we find the blood more or less discolored and changed in constitution, inclosed within the cortical substance, which has become pale and atrophied, and is finally converted into a fibroid layer. *Suppuration* and *induration* are occasionally met with as results of inflammation of these glands. They have been found converted into purulent pouches in the new-born child, and in the fœtus. "*Tubercle* commonly appears in the supra-renal capsules in large masses, and either fuses into pus, inclosed in a callous sac, or is converted into a chalky concretion invested by a fibroid tissue." *Cancer* in the form of encephaloid occasionally attacks the supra-renal capsule, according to Dr. Walshe and M. Roger, only as secondary to disease of the kidney, or (on the right side) of the liver. It causes considerable enlargement, and when hemorrhage takes place the mass may be broken down into a chocolate-colored pulp.

MORBID ANATOMY OF THE URINARY APPARATUS.

CHAPTER XXXV.

Congenital Anomalies.—The kidneys are very rarely absent entirely; but it is not uncommon to find one only. In this case, Rokitansky makes a distinction between the unsymmetrical and the solitary kidney. The former has its usual position and shape, and is only larger than natural. The solitary kidney is produced by the more or less complete fusion of the two organs together; in its lowest degree it constitutes the horseshoe kidney, the lower parts of each being connected by a band of renal substance passing across the vertebral column. In the highest degree, there is only a single disk-like kidney, lying in the median line, and situated much lower down, at the promontory, or in the concavity of the sacrum.

Hyperæmia.—This condition of the kidney is of frequent occurrence, but is almost always the result of some prior general affection, as of obstructive disease of the heart, the scarlatinal poison, or the arrest of the cutaneous transpiration. We shall describe an extreme case, to convey an idea of the state of congestion that may often be inferred on good ground to exist, and shall hereafter refer to it as the commencement of other affections. The kidney is enlarged, its weight often doubled; it is of dark-red color, and drips with blood when cut into. The cortical substance, the medullary cones, the mucous lining of the calyces and pelvis, are all much congested. The former is somewhat softened, of a dark-red color, and presents in many cases small dark-red spots, which are the result of hemorrhage into and between the tubes. The Malpighian tufts are also distinctly seen as minute, reddish, subtransparent grains, prominent on the cut surface. In the medullary cones, the congested vessels form long dark-red streaks. A somewhat turbid sanguineous fluid is contained in the injected calyces and pelvis. The capsule, if the hyperæmia has befallen a healthy kidney, can be peeled off readily. Microscopy shows the Malpighian and other capillaries loaded with blood, extravasation sometimes into the capsule of the former, and often into the channel of the tubes. In a typical case, no other alteration would be visible; but it is scarcely conceivable that the hyperæmia should proceed to any great degree without exudation of fibrinous fluid taking place, which is then seen, having coagulated in the

tubes, forming casts of their interior, and consisting of a granular or homogeneous material entangling blood-globules, and often some detached particles of epithelium. In one case, where we observed the rise of hyperæmia almost from its commencement, and which was further

Fig. 243.

Hemorrhage into Malpighian capsules compressing the tufts.

Fig. 244.

Tube containing some yellow granules, the remains of extravasated blood.

peculiar, in that there was no manifest exciting cause, the fibrinous exudation appeared as extremely pale-reddish, granular films, entangling some renal epithelium; subsequently, casts were observed, and blood-globules. The gradual nature of the commencing effusion was probably the cause that the fibrin did not coagulate at once in the cavity of the tubes. Congestion, then, often reaching an extreme degree, early giving rise to fibrinous exudation, and in many more advanced cases to hemorrhage, and an otherwise sound state of the renal tissue, are the principal features of hyperæmia.

We may here notice somewhat further the circumstance of renal *hemorrhage*. It may occur as an endemic, and, to judge from the absence of bad effects, scarcely serious phenomenon. Dr. Prout remarked its prevalence during the period first subsequent to that in which the cholera prevailed; it was uninfluenced by remedies, and after some weeks ceased of its own accord, and without leaving any unfavorable result. Turpentine and cantharides have not unfrequently caused renal hemorrhage; they act as irritants upon the organ, and produce a state of congestion. Malignant fevers, purpura, and scurvy, are not uncommonly attended with hematuria, and there is good reason to believe that in most cases the blood comes from the kidney. In some of these affections, characterized by a fluid condition of the blood, the casts of the tubes would probably be absent, or very imperfectly formed. Blows on the loins are another cause of renal hemorrhage. In an interesting case recorded by Dr. Johnson, the bleeding recurred several times, the blood coagulating in the tubes, and forming casts which appeared in the urine. A calculus lodged in the calyces or pelvis of the kidney is often the cause of hemorrhage, which, though taking place from the kidney, and perhaps being very abundant, is distinguishable from the preceding forms, as well shown by Dr. Johnson, by the circumstance that there are no casts of the tubes. These cannot be formed; as the blood, not being effused from the renal tissue, but from the mucous membrane,

does not traverse their channels.[1] Cancerous disease of the kidney is sometimes attended with bleeding, but this can scarcely be distinguished from that which is the result of the irritation of a calculus. Blood globules, when retained in the tubes, sometimes undergo change into small yellow corpuscles, very much like those which are often found in the spleen. It is necessary to be aware of this circumstance, as otherwise the observer might suppose that biliary matter was present.

Anæmia.—General anæmia does not seem to affect particularly the condition of the kidney, at least, we have notes of one case of extreme anæmia, the result of menorrhagia, and which at last terminated fatally, in which, although the kidneys were small, the epithelium of the tubes was very perfectly formed. Mr. Simon notices, however, a condition of atrophy of the epithelium, resulting from the obstruction of an arterial branch by atheromatous and fibrinous matter, which would of course produce a local anæmia. In that which is regarded by several as the second stage of renal degeneration, the kidney, though much enlarged, is often remarkably pale. This anæmia, however, is not attended with atrophy, and is itself, probably, in part the result of pressure exercised on the intertubular plexus.

Nephritis, analogous to common inflammation of other parts, and like it often passing into suppuration, is not a very common disease. It is most frequently seen as the result of the irritation of calculi in the kidney, or of inflammation of the bladder, which has either spread up along the ureters, or directly attacked the kidney. Blows on the loins are also mentioned by Drs. Prout and Johnson, as causes of nephritis. The latter also believes that a morbid state of the blood, such as gives rise to carbuncles, may act in this way. Rokitansky speaks of nephritis following acute or chronic diseases, and presenting a type corresponding to the general dyscrasia. Whatever be the cause, the general characters of the inflammatory action will not differ materially, provided it is true nephritis that we are dealing with, and that it passes on to the stage of suppuration. Nephritis, unattended with the formation of pus, would probably be undistinguishable from the condition of hyperæmia above described. In a man, who died in St. Mary's with an enormously-thickened bladder and inflamed kidneys, the bladder being the original seat of disease, and the kidneys secondarily involved, we found the latter organs in the following condition. They were very greatly enlarged, and in some parts very much injected; in others, the red surface had numerous whitish spots, or patches, appearing through it; these were seen in sections to be the ends of long striæ, which commenced at the base of the medullary cones, and extended to the surface of the organ. Between them were interposed streaks of congestion. There was nothing remarkable in the medullary cones, except that they had a coarse aspect. Microscopy showed that the cortical tubes were quite infarcted with their epithelium; in the whitish portions this was especially accumulated, and altered so that it resembled a mass of nuclear particles; the tubes also were not clearly seen; they were doubtless so distended and crowded

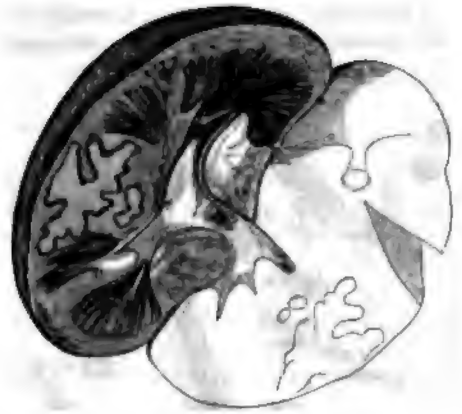
[1] In a case of chylous urine, now under Dr. Chambers in St. Mary's Hospital, a large quantity of blood as well as fibrin comes away in the urine, but there are no casts.

together that their outlines were lost. In some parts the basement-membrane of the tubes was gone, and the contents appeared as a naked strand of nuclei and granular matter, part of which became dispersed between the tubes, and made the mass quite uniform. This mass of altered renal structure was evidently on the point of fusing down into fluid pus. The Malpighian tufts appeared healthy. The medullary tubes were also infarcted, some of them very much, and were opaque, as if containing finely-divided oily matter. There seemed to have occurred also an extra-tubular effusion of plasma, solidifying into a granular stuff. Dr. Johnson, in narrating a case of suppurative nephritis, says, "that the appearances in the kidneys showed the transition from epithelial cells to pus-corpuscles; some tubes being filled with desquamated epithelium, others with pus, while in other parts the pus-cells had accumulated so much as to destroy all trace of tubular structure." The urine in these cases is found to contain cylindrical masses of pus-cells, which have evidently been formed in the renal tubes; this sign, while it lasts, serves to distinguish suppuration in the kidney from that taking place in the bladder, as in the latter case the pus-corpuscles form only shapeless masses. When the suppurating renal tissue is quite broken down, the pus will no longer form casts, the tubes being destroyed. Rokitansky remarks, that enlarging renal abscesses are bordered by a red injected halo, which gives rise to a fusible product, leading to an extension of the abscess. They are always more numerous in the cortical substance, and generally of a roundish shape, while in the tubular substance they are more elongated, like striæ. The mucous membrane of the calices and pelvis, especially when a calculus is lodged in these cavities, is softened and inflamed, and secretes a purulent fluid. The extension of renal abscesses may go on until the whole organ is converted into a mere pouch of pus; in this case, or even before the organ is quite destroyed, the abscess may make its way by the usual process of absorption, penetrating through surrounding indurated tissue, and evacuate its contents in either of the following situations: (1) Externally into the lumbar region; (2) into the cavity of the peritoneum; (3) into the ascending or descending colon, or into the duodenum; (4) into the bronchi after perforation of the diaphragm. Acute inflammation of the kidney may become chronic, or the inflammation may have a chronic character from the outset; its results may be suppuration or induration, and consecutive atrophy of the organ. Dr. Watson thinks that nephritis, confined to the parenchymatous substance, may arise and pass through all its stages without announcing itself by marked local signs, such as pain, &c. This would give the process, though truly acute, an appearance of being chronic. It seems desirable to notice the possible simulation of an abscess by a mass of softened fibrinous exudation, which may be bordered by a red halo as in abscess; the microscope would sufficiently distinguish the one from the other.[1]

[1] We have recently witnessed a very interesting case of fibrinous deposit in the kidneys; in which cavities were formed, closely resembling abscesses. A youth, æt. 20, suffered with symptoms of fever with pus, mucus, and blood in his urine for nine days, at the end of which time he died. The bladder was empty and contracted, and only presented some ecchymosed spots; the left kidney contained several small spots of softening

Degenerative disease of the kidney. Morbus Brightii. Desquamative and non-desquamative nephritis. Subacute inflammation of the kidney.—These names refer to an extremely common and important disease

Fig. 245.



Fibrinous deposits in a granular kidney. The situation of the patch is marked by the irregular outline, which was a deep red.

of the kidney, whose main features were discovered by Dr. Bright, but whose real nature is still a matter of doubt. In calling it a degenerative disease, we have expressed our own opinion respecting it, which coincides very closely with that held by the late Dr. Prout. Our brief limits forbid any detailed discussion, and we shall therefore simply endeavor, first, to describe the morbid alterations which seem to belong to this disease, and then to give some account of its pathology and nature. A kidney in the state of hyperæmia, which we have above described, or somewhat approaching to it, with fibrinous exudation in the tubes, is considered by Frerichs as in the first stage of Bright's disease: we greatly doubt if this is generally the case, and cannot look upon hyperæmia as a *necessary* element in the morbid process. Dr. Johnson considers the same condition as the result of acute desquamative nephritis, and believes that complete recovery may not unfrequently take place. In this we quite agree with him, and so, we think, would most observers. He differs, therefore, from Frerichs, in not considering the hyperæmic condition as constituting the first stage of Bright's disease. The urine produced by kidneys in this state is very characteristic; it is of a rather deep, smoky-red color, with a copious dark-reddish deposit, consisting of blood-corpuscles, renal epithelium, and fibrinous casts entangling more or less of the latter. Sometimes corpuscles are present which resemble those of pus, and like them have compound nuclei. The

fibrin, the right many large ones, several of which had broken down, and given rise to cavities with ragged and sloughy walls. There was no manifest trace of inflammation of the other parts of the kidney; the microscope showed the tubes in a tolerably healthy state. This seems to have been a case of fibrinous deposit confined to the kidneys; it was remarkable that though the kidneys themselves were uninflamed, inflammation had been excited in the peritoneum by the disorganizing processes taking place in the right.

appearance of this deposit will be best understood by reference to the accompanying figure. The hæmatin of the blood-globules exudes from them, and gives the peculiar red color to the urine, which is at the same time scanty and loaded with albumen. It will be manifest that an opportunity rarely occurs of examining kidneys during the existence of the intensity of the hyperæmia, as this has generally subsided, or been removed, before the disease proves fatal; the observer, therefore, must

Fig. 246.

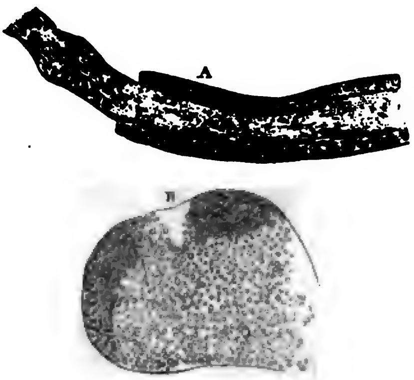
Drawing of red deposit from urine in intense renal hyperæmia.

not be surprised at finding kidneys, which he supposed to have been recently in a state of great congestion, without much appearance of it: the hyperæmia will, of course, decline, in proportion as it is relieved by the hemorrhagic effusion, or by treatment, and the urine, at the same time, will begin to increase in quantity, to lose its red color, and to contain a less amount of albumen. The effusion of serum, with a certain quantity of fibrinous matter, continues for some time after the hemorrhage has ceased, so that casts of the tubes, which become, however, more delicate and pale, traces of renal epithelium and albumen are still discoverable in the urine, until recovery is complete. Crystals of uric acid, in considerable quantity, are also often present during convalescence, and we have also seen oxalate of lime. These may be viewed as indications that the organ is recovering its lost power.

We have thus noticed the hyperæmic or acute inflammatory disease of the kidney, because, though not believing it ourselves to belong to the truly degenerative processes, it produces symptoms in some measure similar, and is regarded by one of the best authorities as always taking the initiative in a more or less marked manner. It may, of course, give rise to a true degeneration, and apparently pass into it; but there is not more connection, we believe, between the two, than between an attack of bronchitis and succeeding pulmonary phthisis. In the first form of renal disease which appears to us truly degenerative, the kidney is considerably enlarged, even more sometimes than in acute hyperæmia; it is generally pale, sometimes of an opaque grayish-white, with mottling

spots or streaks of red; its capsule peels off readily; the surface is smooth and tolerably uniform, or presents opaque spots, indicating the site of future granulations; or these, again, may be just beginning to

Fig. 247.



(A) Tube containing an homogeneous cast, which projects from its broken end. (B) Malpighian body; the capsule is filled with oily matter.

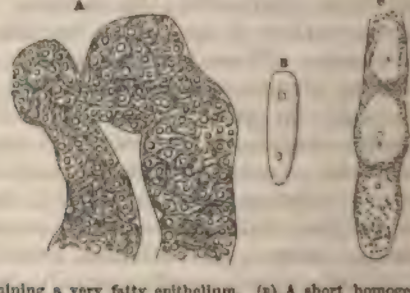
become prominent. The surface of a section shows the thickness of the cortical substance much increased, and the medullary cones also somewhat enlarged and markedly striated, so as to resemble a plume of feathers; they are usually more congested than the cortex. The renal tissue in the latter part appears to be obscured or confused, as if some coagulating fluid had been effused throughout it; it is commonly marked also by similar indications of commencing granulations to those which are seen on the capsular surface. The mucous membrane of the calices and pelvis is somewhat swollen and reddened. The consistence of the kidney is rather diminished; it is rather flabby and soft. The above description applies to many instances of an early degenerative condition; but there are many others also, in an early stage, which differ in several respects. The size of the organ may not be materially increased, though its structure is evidently altered; its consistence may be dense, firm, and brittle, instead of soft and flabby; its capsule may be firmly adherent, and its color more approaching the natural. Cysts are sometimes observable on the surface of the organ, but this is no necessary part of the morbid change. When thin sections are examined microscopically, the following circumstances are observed: The epithelium lining the cortical tubuli is greatly increased in bulk; sometimes its particles are more completely formed than usual, and appear more distinct and separate; sometimes their size is greatly exaggerated, so that a few particles cohering together form a large bulky mass; sometimes the tube is filled with a *mélange* of stunted or withered-looking nuclei, granular matter, and ill-formed celloid particles. The central channel of the tube, which should, normally, be about one-third or one-fourth the whole diameter, is much encroached on and even obstructed by this accumulation of epithelium; from the same cause,

the tube becomes dilated, and hence the bulk of the organ is increased. Coagula of fibrin also are present in some of the tubes, but not in the great majority, and doubtless aid in increasing the obstruction. The

Fig. 248.

(2) Cortical tube, infarcted with epithelium, and bulged in a good part of its extent. (3) Cortical tube containing a dumb-bell crystal of large size. (4) Cortical tube, infarcted by epithelium at (a) below it; some of the separate particles are shown more highly magnified. (5) Bulky epithelium from cortical tube; the group at (a) are remarkably enlarged, those below them are more or less fatty. (6) Medullary tube much infarcted; the contents are seen escaping from the upper end.

Fig. 249.



(A) Cortical tubes, containing a very fatty epithelium. (B) A short homogeneous cast, containing two corpuscles. (C) Portion of a medullary tube, containing three casts, looking much like cysts and oily matter.

The four preceding figures are intended to illustrate the changes observed in the enlarged form of degenerated kidney.

basement-membrane of the tube, in some cases, is natural, in some is decidedly atrophied, and scarcely can be detected. A condition of the epithelium is sometimes observed which is extremely significant, we

Fig. 250.

Microscopic view of epithelium-cells and fibrinous shreds from the tubuli uriniferi of a kidney affected with Bright's disease. (1) Epithelium-cells from the tubuli uriniferi, loaded with oil-globules, magnified 400 diameters. (2) Fibrinous shreds from their interior, having blood-corpuscles and oil-globules entangled in them, magnified 200 diameters. (3) One of the tubuli from a kidney affected with Bright's disease. Oil-globules are seen through its walls.

think, of the nature of the morbid process; it is noticed by Dr. Johnson as peculiar to that form of disease which he denominates non-desquamative nephritis. The epithelium in this does not accumulate and block up the tubes, but appears as a coarsely granular opaque stratum, of the natural width, resting on the basement-membrane.

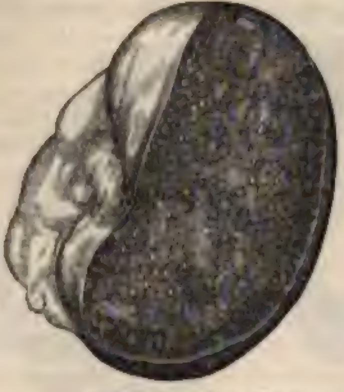
The Malpighian tufts are more opaque than natural, the capillaries being obscured by a film of coagulated fibrin; in some cases this is so abundant, and so mixed with oily matter, that the capsule becomes notably dilated; in others, the pressure of the refluent fluid from the obstructed tube compresses the tuft into a small space at the bottom of the capsule. Oily matter, in the form of minute dark molecules, and various-sized drops, is often present in small, sometimes in considerable quantity; when very abundant, it imparts to the kidney a dead milky or yellowish-white aspect. In such cases, the epithelial particles often become so filled with oily molecules as to resemble very closely granule-cells. In many cases, however, there is no trace of it, and it is certain that it is not of the essence of the disease. In the more advanced instances of degeneration—before, however, atrophy of the kidney has become decidedly apparent, the cortical tubuli exhibit unequivocal traces of breaking up. Their basement-membrane is lost; their epithelium, though still preserving the tubular form, is tending to become a mere detritus, and the commencing appearance of granulations shows that atrophy is taking place in some parts, while others remain prominent and distended by their included cell-growth. Microscopic cysts are sometimes imbedded in the cortical tissue, and may tend, in some measure, to increase the size of the kidney; but they are not so numerous as in the more atrophied condition. The medullary tubuli are less affected than the cortical; those near the base of the cones, especially, often are filled by accumulated epithelium, while those nearer the mam-

mellæ are more free, and contain either some oily matter, or fibrinous casts, or yellow corpuscles, the result of hemorrhagic effusion. The matrix-tissue is little altered in itself, but is often infiltrated with a granulous exudation-matter, containing a few nuclear corpuscles; these are sometimes elongated and developing fibres. The urine produced by kidneys in the foregoing condition is generally paler than natural, of somewhat altered smell, and deposits a whitish sediment, consisting of fibrinous casts, renal epithelium, and vesical scaly particles. Its acidity is less than that of the natural secretion, and its specific gravity, which, in the earlier stages, may be above, tends to fall more and more below the standard of health. While the fibrinous casts contain epithelial particles, it may be inferred that their cell-growth is still produced in the interior of the tubes; but when the urine deposits large cylindrical casts, with very little trace of epithelium imbedded in them, it betokens that the basement-membrane is denuded, and that the condition is becoming more unfavorable. When oil-drops appear in the epithelium or in the casts, it is, of course, a sign that oil is deposited in the kidney; but it is by no means certain that this particular change is of any moment. Blood-globules are not generally present, unless as the result of some recent congestion. Albumen is constantly present, except in a few rare cases, where it disappears for a short time; its quantity varies considerably, being, apparently, much more influenced by other circumstances than by the period of the disease. Frerichs states that the daily drain from the blood varies from 54 to 360 grains. The quantity of all the solids of the urine diminishes as the degeneration advances, the urea, the lithates, the alkaline, and earthy salts are all diminished; in one case, mentioned by Dr. Christison, the total amount of solids excreted from the kidneys, in twenty-four hours, amounted only to $\frac{1}{12}$th of the normal average. Dr. Rees has lately shown that an increased quantity of extractive matter is often poured out in cases of renal degeneration, which constitutes a further drain, in addition to that of the albumen.

The next condition of kidney which we shall describe is often regarded as a further stage of the preceding; this is, we believe, sometimes the case, but not by any means necessarily or universally. The organ is greatly atrophied, and evidently contracted; its surface is covered with irregular prominences, the so-called granulations. Its consistence is considerably increased, so that the structure has, as Frerichs says, a kind of leathery toughness; this is more marked in proportion to the atrophy. The color is decidedly less pale than in the former variety; it often seems to depend very much on a moderate amount of passive hyperæmia. The capsule is always very adherent, but it is worth noticing that, sometimes, when it is thickened, a layer of it may be peeled off, leaving behind an apparently smooth surface. The cortical part is most affected by the atrophy, being reduced, in extreme cases, to a layer two or three lines in thickness; the medullary cones suffer in a less degree. The external form is often remarkably lobulated, reminding one of that of the fœtal kidney. Microscopically examined, the renal tissue is found to have perished extensively, and this generally in proportion to the shrinking of their size. In extreme cases, one may scarce find in a section anything except mere granular

debris; some of these, perhaps, still preserving the contour of the original tube, but the greater part constituting an indefinite shapeless

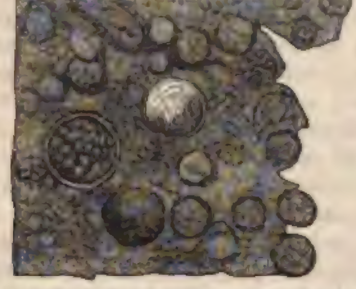
Fig. 251.



Drawing of atrophied kidney.

mass. In other less advanced cases, and in some parts, indeed, of all, the tubes are still discernible: they are irregularly distended, and opaque with granular contents, which have wellnigh, or perhaps com-

Fig. 252.



Cortical part of a very granular kidney, containing very numerous microscopic cysts. The tubes are very much degenerated and broken up. Two Malpighian bodies are shown.

pletely blocked up their canals. Oily molecules, sometimes accumulated in considerable quantity, lie here and there amid the granular matter, and increase the opacity greatly. The granulations are made up of the infarcted convolutions of tubes, and are the parts in which most traces of the natural structure still persist; they remain prominent, because the intervening parts have perished and shrunk in. The basement-membrane of the tubes, in most instances, is lost; occasionally a trace of it may be seen, giving a sharp definition to a mass of epithelium; but, as a rule, it seems to disappear with advancing degeneration, and we cannot confirm the observation of Frerichs and others, that it is still existent among the atrophied tissue.

In specimens where its presence is very manifest in infarcted but not atrophied tubes, we have observed sometimes a single tube with its homogeneous membrane denuded by separation of the epithelium, as

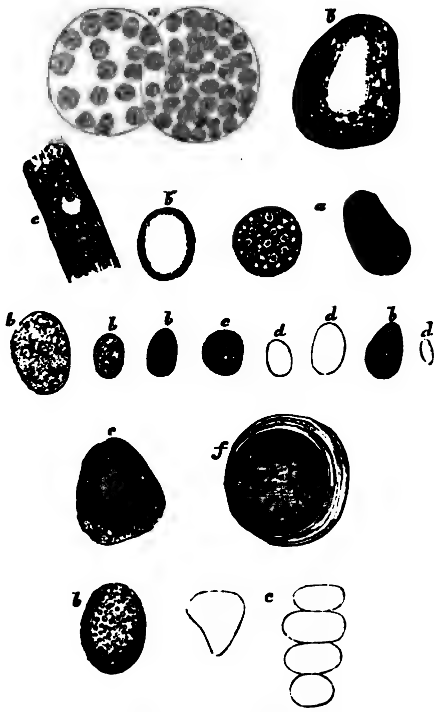
Fig. 253.

The upper figure is from the medullary, the lower from the cortical portion: they are intended to represent a thickened condition of the matrix. Drawn from actual observation by Dr. Sieveking.

noticed by Dr. Johnson. We have also seen what we think has not yet been described, a kind of thickening, or hypertrophy of this membrane, which has seemed to us to result from the atrophy of the epithelium, and its fusion into an homogeneous layer on the inner surface of the tube. The Malpighian tufts, in consequence of the general collapse, appear closer together; a few of them remain tolerably healthy, others are compressed and shrunken; often the capsule is filled, to a greater or less extent, with an oily-looking matter, or the capillaries are obscured by fibrinous exudation. When that peculiar condition of the blood exists, not very uncommon, which leads to the deposit of "bacony matter" in the liver and in the spleen, this is often observed also in the interior of the Malpighian capillaries, and we have not detected it in any other part of the organ. The membrane of the capillaries has rather appeared to us thinned than thickened, as Dr. Johnson describes it, and to this thinned and probably otherwise altered condition of their tunic, we should ascribe the constant draining of serum that takes place. A very important and significant alteration has been observed in the condition of the small arteries by Dr. Johnson; he finds their coats considerably thickened, both the inner of longitudinal and the outer of circular fibres, and he regards this thickening as an instance of true hypertrophy induced by the increased pressure exerted upon their parietes by the retardance of the circulation through the intertubular venous plexus. No particular change is observable in the last-mentioned capillaries, or in the veins, except that the latter often contain firm coagula of blood, which are more or less closely adherent to their walls.

We proceed to the consideration of the cystic growth which often takes place so abundantly in diseased kidneys. These formations appear as vesicles of very various size, varying from microscopic objects of $\frac{1}{1000}$ inch diameter to the magnitude of a cocoa-nut. They contain usually a clear, colorless fluid, which is slightly albuminous, and holds

Fig. 254.



Renal cysts, and cyst-like casts.

(*a*) Cysts containing cells.

(*b*) (*b*) (*b*) (*b*) (*b*) (*b*) Cysts containing granulous matter and nuclei; in *b'*, the celloid contents are disposed so as to form an epithelium round a central space; in *b''*, the nuclei are elongated.

(*c*, *c*) Cysts containing granulous and oily matter.

(*d*, *d*, *d*) Small transparent vesicles.

(*e*) Young cyst diameter, 1-2000th of an inch in the remains of a tube.

(*f*) A cyst with laminated walls.

(*a*) Two doubtful cysts, probably casts, without distinct envelop, consisting of granulous and a little oily matter.

(*b*) An oval fibrinous cast advanced in fatty degeneration.

(*c*) Pale, homogeneous, fibrinous casts.

in solution the ordinary salts of the serum. Sometimes it is of a dark-yellowish color, and more or less viscid, indicating, perhaps, the presence of colloid matter. We found, in one instance, a large quantity of yellowish-granular corpuscles diffused through the fluid, and fragments of an epithelial pavement, consisting of closely opposed nuclei. No urinary principles are found in them, at least as a general rule, but oily matter is not unfrequent, and cholesterin is occasionally present.

As they extend and attain a largish size, they seem to cause absorption of the cortical rather than of the medullary substance, so that on laying open a cyst, a medullary cone may be seen at the bottom of it, and forming perhaps a ridge in its interior. Respecting the origin of cysts, opinions are divided; Drs. Johnson and Frerichs maintain that they are produced by obstruction of the tubes, and subsequent dilatation from secretion taking place within them. Mr. Simon first proposed the idea that they originate as new formations or growths within the tubes, each cyst having its origin in a germ or nucleus particle, such as under healthy conditions might have produced an epithelial cell. Rokitansky and Paget have also adopted this view, and we have ourselves been long convinced of its accuracy. It applies especially to those cases where the cyst-formation is extremely abundant, but we are more inclined to believe that the few and rather large cysts which occur in kidneys not seriously diseased, are produced in the former manner. When the tuft in the interior of a Malpighian capsule is compressed and spoiled, we believe that a cyst may be developed from the capsule in the same way as from a portion of a tube. The smaller cysts contain usually either a clear fluid, or granulous, or an admixture of granulous and oily matter. The larger ones sometimes contain an endogenous cell-growth. The envelop in all is well marked, formed of a distinct homogeneous membrane; it occasionally presents concentric laminæ. Oval and roundish fibrinous casts, when impacted in the medullary tubes, may simulate very nearly the aspect of cysts, especially when some epithelium is imbedded in them. The more or less altered remains of hemorrhagic effusions, in the form of black or yellow matter, are not unfrequently met with in the cortical substance; in some very rare cases small collections of pus have been found, though it is possible these may have proceeded only from disintegrating fibrin; urinary deposits, such as uric acid, urate of soda, and oxalate of lime, have sometimes been observed in the tubes; and lastly, tuberculosis of the kidney has been noticed, when their organs were at the same time affected with Bright's disease. There is nothing very characteristic of the urine passed from small atrophied kidneys; it is generally, however, more abundant, of lower specific gravity, of paler color, less albuminous, and deposits a less quantity of epithelial sediment than that from the enlarged organ. Fibrinous casts will rarely be entirely absent, though their quantity, and the presence or absence of blood-globules, will depend very much on the degree of congestion which may be induced by various circumstances. With regard to the *causes* of renal degeneration, it appears that taking first the predisposing, no age is exempt, but that, as Frerich's Table shows, the greater number of cases occur between twenty and forty. If, however, we exclude the acute hyperæmic attack, as we are inclined to do, the number occurring in the early years of life will be much reduced. The large mottled kidney is often observed in early adolescence, the contracted belongs more to a later period. The male sex seems to be more affected than the female, but we doubt if this would apply to the true degenerations. Whether the scrofulous diathesis occasions special liability to the disease is not quite ascertained; we are inclined to think

that it[1] does; a female suffering from very marked renal degeneration lately told us, that she had lost three brothers and two sisters "by a decay of nature." Occupations attended with much hardship and privation are those in which most cases of Bright's disease occurs. A damp cold climate is most favorable to the production of renal disease, but it is common in tropical regions also. The exciting causes may be referred to two classes, the one tending to induce acute hyperæmia of the kidneys, and to give rise to that form of Morbus Brightii, which is not essentially degenerative; the other by impoverishing the blood, and depressing the general powers, acting as direct promoters of renal degeneration. In the first class we comprise stimulating diuretics, blows on the loins, arrests of perspiration, the congestive influence of the exanthemata, and of cholera. In the second, excess in spirituous liquors, bad nutrition, inveterate syphilis, mercurial cachexia, and exhausting suppurations, as well as the deteriorating effect of continued fever on the general system. We do not think that obstructive diseases of the heart have much to do with the production of renal degeneration, at the most they only act as predisponents. The same may be said of pregnancy; as, though it is, unquestionably, in some instances, the efficient cause of renal congestion, and consequent convulsions from uræmia, yet Frerichs acknowledges that even among such cases traces of real degeneration are rarely discoverable. We regard, therefore, albuminuria in pregnancy and in obstructive heart disease only as an indication that the kidney is the seat of a passive hyperæmia, which we do not think is often followed by actual degeneration. It is possible that the increase of fibrin in the blood, which takes place in the latter months of pregnancy, may, where a predisposition exists, induce true M. Brightii, but this at least is rare. The effects of renal degeneration manifest themselves primarily and principally in the blood, and as we have already spoken of uræmia as a disease of the blood, we shall merely enumerate here the changes which it undergoes. The blood-globules are destroyed to a very great extent, especially in the advanced stages of the disease, the specific gravity and albumen of the serum are diminished, the fibrin remains at the normal figure, the extractive matters are slightly increased, the quantity of oil and of salts is scarcely altered; urea has often been detected in considerable quantity, and becomes most abundant when the secretion of urine is very scanty. As this unhealthy blood circulates through the system, it occasions disorder and disease of different organs. The serous membranes are often inflamed, and pour out copious effusions; the lungs are apt to become congested and infiltrated with fluid, or bronchial catarrh is set up, and becomes habitual; the brain and medulla suffer, and coma, or convulsions, prove fatal; dyspepsia, vomiting, and diarrhœa may announce disorder of the stomach and intestines; chronic rheumatism is a frequent complication; atheromatous disease of the great bloodvessels, and dilatation of the heart, are also with justice attributable in some cases to the poisoned state of the nutrient fluid. The liver is often found affected with more or less of cirrhotic change,

[1] The first form of degeneration in which the kidney is enlarged and pale, or fatty, is often met with in patients dying with tubercles and cavities in their lungs.

but we believe this to be rather a coincident effect of the state of the blood which produces renal degeneration, than a secondary result of that degeneration.

We come now to the consideration of the *nature* of Bright's disease, excluding the acute hyperæmic attack as of a different kind. Frerichs makes it entirely consist in a preceding hyperæmia, occasioning exudation of fibrin into the tubes; this filling of the tubes accounts for the increase of size, and the pallor of the kidney; and the detachment of the epithelium he believes is only occasioned by the fibrinous casts adhering to it, and carrying it along with themselves when they are swept out by the current from the Malpighian tufts. As the coagula and the epithelium are carried forth, the tubes collapse, and the kidney thus passes into a state of atrophy. The causes of the effusion are, according to Frerichs, either mechanical hinderances to the flow of blood in the renal veins, or a supposed paralytic dilatation of the capillaries, which may be directly brought about by irritants, or an unnatural state of the blood, as in scarlatina, or by a reflex action from the skin. We remark upon this theory that it is far too mechanical; that it takes no account of the great number of recoveries which take place after acute hyperæmic attacks, and does not accord with the frequently latent origin of hopeless degenerations; that it does not explain sufficiently the important changes which take place in the epithelium, and in the basement-membrane, nor why there should be so marked a difference between simple Nephritis and M. Brightii. Dr. Johnson's theory, we think, is far more tenable; it is expressed by him as follows: "All changes of structure commence in the secreting cells of the gland, and are the result of an effort made by the cells to eliminate from the blood some abnormal products, some materials which do not naturally enter into the composition of the renal secretion." With the first part of this statement we quite agree, the second we think, is much more doubtful. That a diseased state of the blood is the essential cause of renal degeneration, we have little doubt, but we conceive that this consists in an unnatural state of some of the normal constituents of the blood, probably the fibrin or albumen, and that this induces an unhealthy nutrition of the renal tissues. As we are ignorant of the way in which the epithelium subserves the secretory process, how it eliminates the urea, uric acid, &c., from the blood in which they are formed, we cannot understand in what way a diseased state of the epithelium disables it from fulfiling its function. Comparing, however, generally, the healthy with the morbid structure, it may be said that the former conveys the idea of a delicate, quickly forming, and quickly changing material, similar to the gray matter of the hemispheres, while the latter appearing coarser, more granular, and having often more shaped and more consistent particles, seem certainly much less apt to undergo rapid change. The hypertrophy of the epithelial particles, and their accumulation in the cavity of the tubes, may be only the result of their not disintegrating in the formation of the secretion as they normally should. Some time ago we examined an enlarged thyroid, in which most of the vesicles were filled and distended by collected secretion, but some were filled with epithelium only. In the same way, sebaceous follicles distended into cysts sometimes contain

considerable quantities of epithelium, sometimes oily matter preponderates. The accumulation, therefore, of epithelium within the tube, is no sign that the cells are striving more actively to eliminate morbid matters, but only that, being unhealthily nourished, they do not disintegrate in the secreting act as they normally should. The deposition of oil in the cells seems to us also an indication that their vitality is at a low ebb, that the plasma out of which they are formed does not maintain its normal composition. So also, the perishing of the basement-membrane seems strongly to indicate unhealthy nutrition. The explanation given by Dr. Johnson, of the mode in which hyperæmia of the kidney and consequent exudation is produced, seems to us most consonant with sound pathology. He starts from the important experiment of Dr. Reid, which shows so well the effect of arrested nutrition in obstructing the passage of blood through the capillaries of a part. When the air-cells of the lung contain a normal proportion of oxygen, the blood traverses freely the pulmonic capillaries; when they become surcharged with carbonic acid, the current of the blood is obstructed in the same capillaries. The increase of pressure in a hæma-dynamometer in an artery, and the decrease in a corresponding vein, show that the unaerated blood cannot traverse freely the systemic capillaries. As the cells lose their power of eliminating the urinary constituents, the blood passing through the tubular venous plexus is retarded in its course, and a continually increasing congestion is set up. This affects necessarily the Malpighian tufts, and occasions the draining off of liquor sanguinis from them, and often hemorrhage also. The thickening of the walls of the small arteries before mentioned is another result of this backward pressure. We think the term "chronic desquamative nephritis" unsuitable, as the hyperæmia which it induces is only of a passive kind, resulting from obstruction, and herein probably essentially differing from the active condition of the acute hyperæmic attack. Deposition of oil in the degenerating epithelium we hold to be accidental, and not in any way essentially modifying the morbid state. As it is most common and abundant in the renal cells of dogs and cats, in England and Germany, without any disease, so its presence in the condition we are considering cannot be of much moment. With regard to the two forms of diseased kidney which we have described, we feel some degree of doubt as to the exact relationship they bear to each other; some regard the atrophied kidney as the more advanced condition of the enlarged one, others consider the two as distinct varieties. We strongly incline to the latter opinion, and to the belief that the enlarged degenerated kidney possesses pathological affinities with phthisis and the class of scrofulous maladies, while the contracted eminently granular kidney is allied to such changes as those seen in cirrhosis of the liver, and contraction and thickening of the cardiac valves. Between the two forms, of which we have tried to give a typical description, of course there are very numerous intermediate ones which the student must expect to find. In some of them, it is scarcely possible to determine by the naked eye whether the organ be diseased or not; but a careful microscopic examination of the cortical tubes, the Malpighian bodies, and the medullary tubes, together with a reference to the state of the urine during life, will usually clear up all

doubt. The adhesion of the capsule to the surface, if decidedly unnatural, is a valuable morbid sign.

Tubercular disease of the kidney.—Tubercle is not of frequent occurrence in the kidneys. These organs stand only in the eighth place of the scale given by Rokitansky. It is found sometimes in the miliary form, sometimes in larger masses, which preserve the shape of the part in which their substance seems to be infiltrated; sometimes it extends to the kidneys, from the mucous membrane of the calices, and proceeding up along the sides of the cones, gradually advances inwards into the secreting structure. The miliary granulation is sometimes (when a high degree of tubercular dyscrasia exists) associated with a considerable amount of hyperæmia of the organ; when the deposit takes place in a chronic manner, the surrounding tissue is quite pale. The large masses that extend throughout the diameter of the kidney, from the surface to the hilus, are remarkably bloodless. This is well shown in some injected specimens in the museum of St. George's Hospital. When the tubercular deposit extends to the renal tissue, from the mucous membrane of the calices and pelvis, these cavities become remarkably enlarged; they extend up on the sides of the cones, into the cortical substance, and approach more or less near to the surface. At the same time, the whole organ is enlarged, and appears rather pale. The epithelial lining of the tubes is more or less opaque and granular, or of oily aspect. As in other situations, the tuberculous matter tends to soften and break down, and thus cavities are formed sometimes of rather large size. These may contain a mixture of tuberculous detritus and pus. Fibrinous moulds in great numbers are sometimes present in the tubes. The middle period of life is that at which the disease is most liable to occur. In most cases there is a deposit of tubercle in other organs, especially in the lungs, and often in various parts of the genito-urinary apparatus.

Cancer.—We have not exact data for determining the frequency of renal cancer, but it is certainly not rare. Secondary seems to be more frequent than primary cancer. Scirrhus is rarely if ever found; and the same may be said of colloid. Encephaloid growths, especially, we think, in children, attain in the kidney an enormous size. A case has been mentioned to us by Dr. T. K. Chambers, in which the weight of the tumor was three-fourths that of the whole body. No trace of the kidney was discoverable in this case, on the side of the tumor, and the ureter was lost in it above. The liver contained some smaller tumors of similar kind. These facts seem sufficiently to prove the renal origin of the tumor. In a specimen in the museum of St. George's Hospital, there are several small cystic cavities in a carcinomatous renal growth. The statement of M. Rayer, that cancer of the liver and right kidney frequently coexist, as well as cancer of the adjacent parts of the stomach, or descending colon and left kidney, is confirmed by Dr. Walshe. Rokitansky notices that cancer of the kidney often coexists with cancer of the testis on the same side; the renal disease, we think, is most commonly developed after that of the testis. "In thirty-five cases of renal cancer, the disease," Dr. Walshe says, "affected both organs sixteen times, the right alone thirteen times, the left alone six." The table given by this author, shows that the period from fifty to seventy is that which is far

most liable to cancer of the kidney. Ten cases only occurred in the previous years of life, while nineteen were noted in the succeeding twenty.

The urine excreted by cancerous kidneys, may long retain its natural characters. In a case recorded in the Transactions of the Pathological Society, 1846–47, nothing remarkable seems to have been observed in it, although both kidneys were converted entirely into encephaloid masses. When, however, the growth softens and breaks down, blood, puriform matter, or cancerous detritus, may appear in the urine.

Entozoa occurring in the kidney, are the acephalocyst, the cysticercus, and the strongylus gigas. The so-called hydatids from the first of these have, in rare cases, been passed with the urine.

The *adipose tissue*, in which the kidney lies imbedded, may increase to such a degree as to penetrate by the hilus into the substance of the organ, impede its nutrition, and induce a kind of atrophy. Rokitansky states that, in the highest degree of this change, the kidney presents the appearance of a mere mass of fat, without the slightest traces of renal organization; the urinary passages at the same time being atrophied and obliterated.

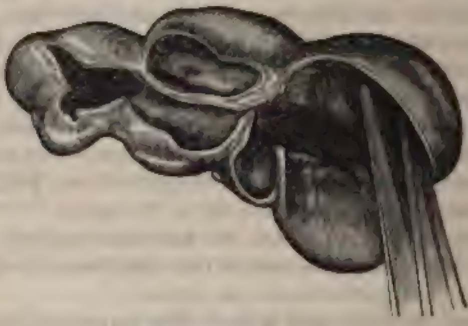
The *capsule* of the kidney may be inflamed, in consequence of which fibroid thickening may take place, and more or less of induration, atrophy, and obliteration of the organ. The cortical substance is especially apt to be involved, and the surface is sometimes overspread with purulent matter, while the tissue itself becomes sloughy or gangrenous, or is only congested and softened.

ANOMALOUS CONDITIONS OF THE URINARY PASSAGES.

Under these we comprise the ureters, and their upper termination, as the pelvis and calices of the kidney. The ureters may terminate from congenital defect, in a cul-de-sac, either in the vicinity of the kidney or of the bladder. Sometimes they are double or triple, usually from fissure of the pelvis of the kidneys, but they generally unite again before their vesical termination. It is not uncommon to find them considerably *dilated*, when the opening into the bladder has been greatly narrowed or obliterated. The calices expand at the expense of the renal tissue, and extend outwards towards the surface, till at length there remains only a thin layer of the cortical substance compressed against the investing capsule, and the kidney is converted into a number of pouches, separated by membranous loculi, which contain the remains of the medullary cones. The surface of the kidney becomes lobulated in a marked manner from the pouches, pressing outward between the interlobular septa. The ureters are at the same time distended, sometimes to that extent that they resemble a portion of small intestine; at the same time, their walls are somewhat thickened, so that they do not appear to be much thinner than natural; they only attain, however, a considerable thickness when there is concurrent inflammation. The ureters become also increased in length, and therefore do not lie straight, but are thrown into coils or flexures. Their mucous lining does not appear to be so often inflamed or ulcerated as that of the calices and pelvis. The

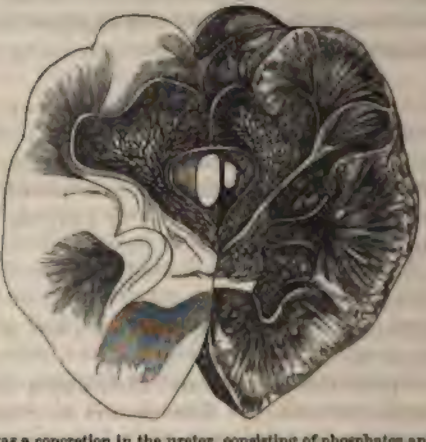
pouches formed by the dilatations of these, are often filled with puriform fluid, or with a mixture of pus and urine, or even with clear serum only. To the latter condition, the term *hydrops renalis* has been given. It seems to take place when the obstruction to the flow of urine into the bladder is complete, and when, in consequence, after extreme distension

Fig. 255.



Kidney converted into cysts.

Fig. 256.



Pyelitis; there was a concretion in the ureter, consisting of phosphates and animal matter.

and atrophy of the renal tissue, the secretion of true urine ceases, and is replaced by a mere serous fluid. After a time, such tumors may diminish, and almost disappear, from the absorption of their contents. The ureters in such cases also contract, and become obliterated. It frequently happens that the renal tissue not only atrophies, but also becomes inflamed, and infiltrated with pus, so that it is still further incapacitated from carrying on its function, and death ensues from the effects of urea being retained in the blood.

Inflammation of the urinary passages often coexist with the state of dilatation; it may also occur, though much more rarely, as a primary disease. It is not unfrequently produced by the irritation of calculi, or

results from the extension of vesical disease, or from metastasis of this, according to Rokitansky. Stricture of the urethra is a common cause of disease of the mucous membrane of the bladder, as well as of dilatation of the ureters. The inflammation set up there is very prone to creep backward, and affect the urinary passages, which are predisposed to it by their unnatural distension, and the prolonged contact of unhealthy urine. The mucous membrane is found in various degrees tumefied, injected, or of a saturated red color, of villous aspect, and covered with a muco-purulent fluid. Perforation of the ureters may take place in consequence of sloughing, the urine infiltrating into the adjacent tissues, and producing either extensive sloughing, or circumscribed abscesses. The inflamed mucous membrane, in many cases, either secretes phosphates, or a mixture of these and carbonates; or these may be deposited from the urine. When atrophy of the kidney takes place, these saline deposits, cemented together by mucus, form a "yellowish-white, greasy, and chalky pulp, which fills the calices," and is inclosed by the wasted organ as by a cyst. In other cases, a renal calculus, such as is described by Dr. Prout as the Phosphatic, may be produced in this way, and become itself a further cause of irritation and disorganization to the kidney.

Rokitansky mentions the occurrence of inflammation, which he distinguishes by the name Exudative, from the above catarrhal form, as a secondary affection in cases of serious blood disease. It is observed in typhus, in the exanthemata, in diphtheritis, and acute tuberculosis, and also in pyæmia, and occasions the formation of unhealthy fibrinous effusions upon the mucous surface, associated in cases of necræmia with hemorrhage.

Cysts, containing a glutinous or hard (colloid ?) matter, about the size of millet-seeds or peas, are occasionally found developed under the mucous membrane of the urinary passages.

Tubercle.—Rokitansky states that this "is always a symptom of tubercular disease, that has spread from the male genitals to the urinary organs." We think some cases recorded in the Transactions of the Pathological Society, and some that we have seen ourselves, show that this is by no means necessarily the case. It is most frequent in the ureters when the kidneys are involved at the same time, but we have seen it in them when the kidneys were healthy. Usually, there exists at the same time tuberculosis of some important organ, as of the lungs or the hip-joint. The deposit takes place in the submucous tissue, and forms, when its progress is chronic, gray granulations, which become yellow, soften, "and give rise to small circular ulcers." When the disease is more acute, larger patches of deposit are formed, or "the mucous membrane becomes infiltrated throughout with the tubercular product of inflammation, which is at once detached as a cheesy, purulent mass."

Cancer but rarely attacks the urinary passages, and only when it is elsewhere in process of development. The disease may extend to them from the kidney, from the lumbar glands, or from the bladder, either when it is itself primarily affected, or involved in uterine cancer.

ANOMALOUS CONDITIONS OF THE BLADDER.

This receptacle is subject to various *congenital malformations*, of which we shall only mention two. The first is termed inversion of the bladder, and results from a defect in the lower part of the abdominal parietes. There appears in the hypogastrium "a red, mucous, dilated

Fig. 257.

Extrophy of the bladder. *a*. Everted bladder. *b*, *b*. Orifices of the ureters. *c*. Penis without urethra. *d*, *d*. Pubic symphysis. *e*. Scrotum and testis. *f*. Congenital inguinal hernia.—From Gross on the Urinary Organs.

spot, the edges of which join with the common integument; in the male sex it passes downwards, so as to terminate in the fissure of the urethra; in the female it is surrounded by two diverging tumors, which represent the labia, and it terminates in the lamina of the general integument which invests the rima vulvæ." The opening of the ureters are seen at the lower and lateral parts of this mucous surface, which is, of course, the posterior wall of the bladder. In a case which we saw recently, the penis was very short, and the canal of the urethra open above in its whole length. The second malformation is attended with fissure of the opposite side of the bladder, and of the adjacent cavities, so that a kind of cloaca is formed, similar to that which exists in the lower animals. In some rare cases the urachus remains pervious, so that when urine is passed it escapes at the umbilicus. In others, again, the bladder has no external opening, the communication with the urethra is not formed.

Dilatation of the bladder is no uncommon occurrence, and may be occasioned either by paralysis of the muscular tunic, or by some obstacle to the outflow of the urine, as stricture of the urethra. We think that the amount of dilatation is greatest when the muscular coat is paralyzed,

and that in the other class of cases, where some obstruction exists, the great hypertrophy of the muscular fibres which is induced prevents the distension becoming so great. Rokitansky, however, seems to consider obstruction as the most powerful cause of dilatation of the bladder. The effect of the stronger contractile coat of the bladder in preventing dilatation, is shown in some cases recorded in the Transactions of the Pathological Society, in which it is mentioned that the ureters were much distended, while the bladder was contracted, or not dilated. The bladder may be so dilated as to rise above the umbilicus considerably, and to contain twenty pints of urine. The paralytic dilatation depends, we believe, in some cases, on fatty degeneration of the muscular coat. Diverticula, or partial dilatations of the bladder, are not infrequent. They are always found in cases in which the muscular tunic is hypertrophied, and seem to be produced by protrusions of the mucous membrane taking place between the fasciculi, which are subsequently pushed outwards more and more by the pressure of the urine. The lateral

Fig. 258.

Sacculation or partial dilatations of the bladder; section of the bladder and prostate. *a.* Mucous surface of the bladder. *b, b.* Lateral lobes of the prostate. *c.* Middle lobe. *d.* Large cyst or pouch, partially laid open, and communicating with the bladder by a small orifice.—From Gross on the Urinary Organs.

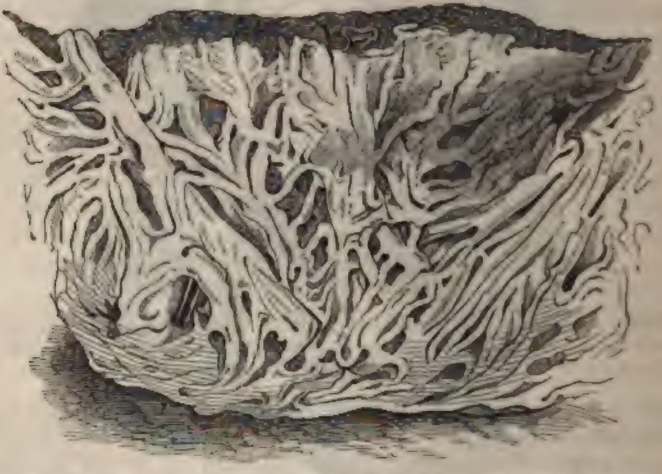
portions, the posterior surface, or the neighborhood of the fundus, are the situations in which diverticuli usually form. They have no muscular tunic, except, occasionally, a few scattered fibres, which Rokitansky suggests may be some evidence of their being congenital. Calculi get into these pouches sometimes, and become so lodged and concealed as to escape detection by the sound.

Contraction of the bladder most often is rather apparent than real, and depends on irritation of the mucous lining, with hypertrophy of the muscular coat. Sometimes it is partial, or may cause a kind of hour-glass constriction of the cavity. When a calculus is present, the

walls are sometimes found closely embracing it; and a case is mentioned by Morgagni, in which the bladder was so closely contracted around a needle, that there was scarce room for anything more in its cavity.

Hypertrophy of the muscular coat is observed in cases where that tissue is unusually exercised, and is often of a manifestly beneficial tendency. When the mucous lining is irritated by the contact of unhealthy urine, perhaps in some degree inflamed or ulcerated, the reflected stimulus from the spinal cord becomes more intense, and the contractions of the muscular fibres more energetic. Again, when in consequence of stricture, the difficulty of expelling the urine becomes great, the contractile force is increased to meet it, and this increase is occasioned by the greater exertions which are necessary. The appearance of the inner surface of a bladder whose muscular coat is hypertrophied, is peculiar, and is well compared by Rokitansky to that of the right ventricle of the heart. The muscular fasciculi become unusually prominent, and by their divisions and interlacements produce a kind of irregular network into

Fig. 259.



Hypertrophy of the muscular coat of the bladder.—From Gross on the Urinary Organs.

the meshes of which the mucous membrane dips, and through which it may be forced in sacculi. The technical term for this condition is *columnated*, or in the French original, *vessie à colonnes*. Dr. Walshe has seen polypoid growths from the inner surface of the bladder, consisting of prolongations of the mucous and submucous tissue, in a state of simple hypertrophy. If we except these, and the diverticula before mentioned, it does not appear that there is any true hypertrophy of the mucous lining. Rokitansky has, in rare cases, seen the mucous membrane *atrophied*, "reduced to a very delicate, shining membrane, resembling the arachnoid," while the muscular coat almost entirely disappears.

The bladder is liable to various *displacements*. It may form the contents of inguinal, vaginal, and perineal hernia, it may be introverted and forced into the urethra, and even in females project from the meatus

urinarius externally.[1] When hernia of the bladder occurs, it is either in part, or, more rarely, completely destitute of peritoneal covering. This depends on the anterior part of the viscus, which has no serous covering, being the first to prolapse; but, as the organ descends, the posterior part which is lined, carries with it the peritoneum, and thus forms a sac, into which intestine or omentum is often protruded.

Hyperæmia of the bladder of a passive kind, occurs when there is some obstruction to the free passage of blood through the pelvic veins and the V. cava; it is, therefore, associated generally with a similar condition of the adjacent viscera. It produces increased secretion of mucus, sometimes spots of extravasation, and occasionally such dilatations of the veins as have been termed vesical hæmorrhoids. These, however, do not appear like the common little tumors of the rectum, but rather as prominent and distended vessels.

Inflammation of the bladder is much more often seen in a chronic than in an acute form; this depends partly on the acute stage of the disease in recent cases having generally subsided before death occurs, partly on the greater frequency of cystitis, which is chronic from the commencement. The appearances in *acute cystitis* are strong vascular injection of the mucous lining, with brownish patches in the vicinity of the neck and fundus; more or less thickening of the membrane, with exudation of fibrin or pus on the surface, or foci of the latter in its substance. The mucous tissue may be ulcerated at several points, softened, or affected by commencing gangrene. Abscesses may form in the substance of the parietes, and open either into the cavity of the bladder, or upon its external surface. Sometimes the mucous membrane is almost completely destroyed, a few shreds or filaments being the only traces remaining, while the muscular tunic is left as if cleanly dissected. This is probably the result of phagedenic ulceration.

Chronic cystitis may be the condition resulting from one or more attacks of the acute form, or may be produced by the extension of urethral inflammation, or by the irritation of unhealthy urine, or of calculi. Its characters are various degrees of vascular injection, mingled with dark-reddish, slaty or bluish-black discoloration, more or less tumefaction of the mucous membrane, with secretion of mucus, or muco-pus, often in considerable quantity. Sometimes, from the irritation excited, the muscular coat becomes hypertrophied and columnated; but the more ordinary condition in chronic cystitis, is the thickening and more or less uniform induration of the parietes, which assume an homogeneous, lardaceous appearance, doubtless from their infiltration with exudation matter. It not unfrequently happens that an acute attack, or exacerbation, supervenes upon a state of chronic inflammation. The following abridged account of the appearances which then present themselves, is taken from Rokitansky: "The bladder is found dilated, and filled with decomposed, intensely alkaline urine, mixed up with blood of a brown color, viscid mucus and pus, sanies, lymph, and detached portions of

[1] In the Report of the Path. Soc. 1852–53, there is an instance recorded by Mr. Pilcher, in which about two-thirds of the bladder were extruded from the abdomen through the inguinal canal, and lodged in the scrotum. The hernial portion was large enough to contain 50 oz. of fluid.

mucous tissue, in the shape of discolored flocculi, or larger patches." The mucous membrane, incrusted by a deposit of amorphous and crystalline phosphates, is sometimes "of a dark-red color, appears spongy, softened, and pultaceous, is easily detached and bleeds; when chocolate-colored or greenish, it is found purulent, infiltrated with sanious matter, or converted into a friable flocculent tissue, which is traversed by the urinary sediment." In some cases, the submucous and muscular tunics are exposed, and are in various stages of softening, suppuration, and disintegration. As the morbid action advances outwards, the peritoneum at last becomes involved, and general inflammation of this membrane may be set up. Ulceration, attended or not with suppuration, sometimes extends deeply and gradually, and at last perforates the walls, when extravasation of urine takes place, if not prevented by inflammatory exudation, and adhesion of adjacent parts. The further progress of ulceration sometimes forms a communication with the cavities of the adherent viscera; in this way the walls of the rectum, the colon, and the ilium, have been perforated, and their contents have made their way into the cavity of the bladder. We lately examined a portion of a bladder which had long been the seat of calculous irritation, and had been further inflamed by the operation for lithotrity, the man dying of pyæmia. The true mucous membrane was in great measure destroyed; there was no trace of basement-membrane or epithelium; the tissue was thickened by indurating exudation and granular matter, and incrusted with amorphous granular particles and prisms of triple phosphate; or scraping off this layer, the surface beneath appeared red and bleeding. No trace could be found of the muscular coat, it was replaced by a thick layer of fat, consisting of large, well-formed vesicles. Chronic or subacute inflammation of the bladder is very commonly an attendant upon paraplegia, and proves the immediate cause of death. The inflammation is set up, we conceive, in the same way as that of the eye is when the fifth pair of nerves has been divided, and results from loss of the nutrient power of the tissues, and consequent stagnation of blood in toneless vessels. At the same time, the urine rendered alkaline by the decomposing influence of the vesical mucus upon the urea, reacts, no doubt, upon the inflamed membrane as a further cause of irritation. The urine is turbid with quantities of muco-pus and detached epithelium, contains often albumen, sometimes blood, and always prisms of the triple phosphate. The coats of the bladder undergo similar changes to those above mentioned, but of a marked asthenic character. The mucous membrane is congested, and thickened and altered by fibrinous exudation, or purulent, or sanious; it is incrusted by a phosphatic deposit, and in parts may be gangrenous. The muscular coat is also more or less affected, and the submucous tissue. Rokitansky describes *exudative processes* of a croupy kind, as not very unfrequent in the bladder; they occur in the course of exanthematic diseases, in pyæmia and typhus. The exudation does not affect generally a large surface, but is limited "to round spots or striæ." The mucous membrane beneath the exudation is more or less injected, tumefied, and indurated, or in processes of lower character is softened and converted into a pulpy, gelatinous, sanious, or purulent mass, or even becomes gangrenous. Rokitansky has not seen variolous

pustules upon the vesical mucous membrane, as others state they have, but he mentions, what we have once observed ourselves, an eruption of minute miliary vesicles, containing a clear serosity upon the surface; they accompany, he says, catarrhal inflammation and slight exudative processes, as well as Asiatic cholera. Acute and chronic inflammation of the muscular coat of the bladder are both spoken and written of, but the former appears to take place only as a part of general cystitis, and the latter, if it intend more than hypertrophy of the muscular fasciculi, is only that general infiltration of the parietes, with induration-matter, which we have before noticed.

Pericystitis, however, seems to be a more distinct affection; it consists in the spontaneous inflammation of the cellular tissue surrounding the bladder, arising either as a primary or a secondary process. It is to be regarded, Rokitansky says, as a localization of pyæmia. From its original seat, it is apt to spread to the areolar tissue round the rectum, to the anus, and into the scrotum; it may involve also the coats of the bladder, and cause perforation of them. It is sometimes of a chronic form, and then gives rise to induration, rigidity, and callosity of the bladder.

Softening of the mucous membrane, not resulting from inflammation, was observed by M. Louis only twice out of five hundred autopsies; in these, the tissue was converted into a kind of pale mucilage. Rokitansky has seen it only once, in a case of typhus.

Tubercle is infrequent in the bladder, and is sometimes absent when the kidneys are extensively affected by its deposit. It is only met with in the form of separate granulations, which are surrounded by more or less hyperæmia, according to the rapidity of their production; these soften and give rise to circular ulcers of the mucous membrane covering them. The cervix and fundus are the parts chiefly affected.

Cancer is much more often seen in the bladder as the extension of disease from contiguous parts than as the primary phenomenon. This at least seems to be the more general opinion; but Dr. Walshe affirms that primary vesical cancer is far from being so uncommon as is generally supposed, and we are quite inclined to agree with him. Scirrhus is very rare in the vesical parietes. Mr. Coulson has never seen it, nor has Sir B. Brodie, except where it constituted part only of a morbid growth. Rokitansky mentions having seen it extending over large surfaces of the sides of the bladder. Encephaloid, forming nodulated prominences or cauliflower-like excrescences, is the form which vesical cancer commonly assumes. These may be of very various consistence, and often very vascular, easily bleeding, and situated especially at the trigone, the neck, the fundus, and the vicinity of the urethral orifices. They are developed in the submucous tissue; but, as they grow, the mucous membrane is destroyed, and either an ulcer is produced, or a soft, luxuriant, fungous mass. They produce irritation of the bladder, more or less difficulty in micturition, and, in the latter stages, hemorrhage, which may be considerable and difficult to arrest. The urine contains mucus, sometimes blood, cancerous detritus, and portions of encephaloid matter, at various times, when they happen to become detached.

MORBID CONDITIONS OF THE URETHRA.

We notice the following malformations: fissure on the upper surface (*epispadia*), or on the lower (*hypospadia*); the former, when extending the whole length, occurs as a complication of eversion of the bladder; the latter accompanies fissures of the scrotum, and occasions a resemblance to the female conformation. The urethra may terminate at various points of its normal course, in the perineum, the root of the scrotum, or anywhere between this and the glans; the opening in these unnatural situations is very small, and sometimes is completely closed (*atresia urethræ*). A kind of cloacal formation may also be produced by the urethra terminating in the rectum, or in the female in the vagina. The diameter of the canal may be congenitally narrowed at the extremity, or at other parts.

Contraction, however, is much more commonly the result of inflammatory disease, under which head we shall describe it.

Dilatation of the urethra is often produced by some obstruction to the flow of urine; it occurs, for the most part, in the membranous portion, which is expanded into a pouch, sometimes as large as a small orange. The mucous lining of these pouches is usually "injected and thickened, presenting fungous vegetations, and occasionally coated with lymph." The urethra is frequently *distorted* from its normal direction, either by the dragging of large scrotal herniæ or hydroceles, or by the pressure of tumors. Enlargement of the lateral lobes of the prostate pushes it to one side, of the middle lobe divides it into two passages. The length of the canal, in such cases, is increased.

Lacerations of the urethra may be produced by mechanical injuries, by the passage of fragments of calculi, or by ulcerative destruction. They often give rise to urinary fistulæ.

Inflammation of the urethra, of the catarrhal kind, is exceedingly common, and constitutes the misnamed gonorrhœa. It commences at the anterior extremity, and gradually proceeds backwards, in very severe cases extending even to the bladder. The mucous lining becomes swollen, injected, and covered with mucous or muco-purulent secretion. Its follicles and lacunæ are attacked, especially the lacuna magna; in the chronic state they are enlarged and relaxed, and pour out, as well as the general surface, a thin mucous, so-called gleety, discharge. During the acute stage, when the inflammation extends deeper to the fibrous structure of the corpus spongiosum, exudation of fibrin sometimes takes place in the venous cells, which renders them incapable of distension, and thus occasions, during erection, a bending of the penis towards the affected part which is termed *chordee*. Abscess may form also in the same situation, from suppuration of the exuded fibrin, or, perhaps, also from severe inflammation of the lacunæ. The inflammation may spread along the continuous mucous lining to other adjacent parts, to Cowper's gland, the prostate, the vesiculæ seminales, and the testicles. This extension of the morbid action to other parts is commonly attended by a subsidence of it in its original seat, so that it is often questionable whether actual metastasis has not occurred. The gonorrhœal discharge passes through

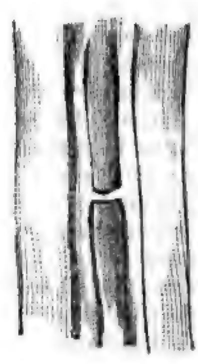
the same stages as that from other inflamed mucous surfaces; it is at first a thin mucous fluid, then more tenacious and muco-purulent or purulent, and, as the inflammation subsides, it becomes again thin and pale. When a chancre coexists with gonorrhœa, "the discharge has usually a grayish or reddish tint, or sanious aspect." The mucous follicles in the vicinity of the meatus are liable to be specially affected both in the male and female. Dr. Oldham appears to refer to this condition under the name of follicular inflammation of the vulva; and Kleeberg, as quoted by Dr. Adams, thus speaks of their condition in the male: "The orifices of the lacunæ become closed by inflammation, and in the course of two or three days pustules are formed in their places, which break and discharge a yellow pus. The orifices of the large mucous follicles are now seen dilated and surrounded by a swollen dark-red border, and they discharge a muco-purulent fluid into the urethra." The disease sometimes assumes a chronic form. The contact of unhealthy vaginal secretion is the most common cause of urethritis, and it is important to be aware that this effect may be produced by the fluids of females who are perfectly chaste. Stimulating injections, the irritation of calculous fragments, the presence of stimulating diuretics in the blood, the materies morbi of gout, of influenza, and the suppression of cutaneous eruptions, are mentioned as causes of this inflammation. Abrasions and excoriations of the urethral mucous lining are occasionally found when it is inflamed, but ulcerations are (probably) always the effect of other causes. The syphilitic poison producing urethral chancre, the presence of foreign bodies, calculi, &c., the irritation caused by a stricture in the part behind, softening tubercle, are so many causes of more or less extensive ulceration. It is said by Mr. Adams to occur in rare cases spontaneously.

A very frequent result of inflammation is *stricture*. This consists in a narrowing of the canal from some organic change in the structure

Fig. 260.

Strictured urethra.

Fig. 261.



Strictured urethra.

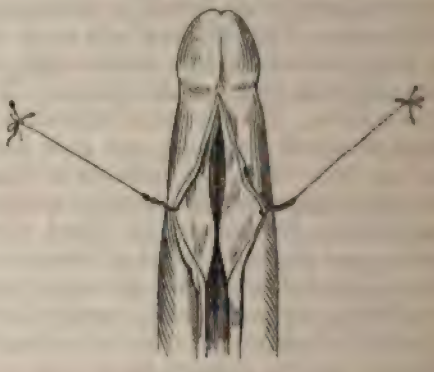
of the part itself, or in that of those around. It may affect any part, but is most frequent in the anterior part of the membranous portion. Out of one hundred and eighty-nine cases examined by Mr. Phillips,

the seat of the stricture was in one hundred and thirty-eight from four to six and a half inches distant from the meatus. Contusions and wounds occasion stricture of the urethra, which, in severe cases of the latter is extremely intractable. The simplest form of stricture is when the canal is partially occluded by a fold of membrane passing across it; this may be of such a shape that a crescentic, or, sometimes, an annular opening is left. Several of these strictures may coexist in the same urethra; as many as eight are said to have been observed by

Fig. 262.

Stricturecl urethra.

Fig. 263.



Strictured urethra.

Calot. They are probably produced by the organization of fibrin effused on the mucous surface; others believe that they may result from the healing of an ulcer, or the raising up of a fold of the lining membrane. In the more common kind of stricture the urethra is narrowed in a much greater extent of its course, and sometimes in an extreme degree. Half an inch or an inch is not uncommonly the length of the contracted part, and sometimes the whole extent of the spongy portion is affected. The stricture occupies sometimes one side, at others it completely encircles the canal. It is not difficult to understand the mode of its production, which is very illustrative of the general contractile tendency of exudation-matter. Fibrin is effused during inflammation in the mucous tissue itself, or in the submucous, as well as sometimes in the corpus spongiosum; if this be not absorbed, it passes into the state of fibroid or induration-matter, and continually tends to shrink up and contract into a narrower space. In proportion as this takes place, the canal must be contracted. The mucous membrane lining the indurated part, is often ulcerated and destroyed, commonly from the mechanical effect of catheters pushed against it; but it may also take place spontaneously, and it has happened that the indurated part being destroyed by the extending ulceration, the stricture has thereby been cured. A more common and less favorable result of deep ulcerations is the perforation of the canal and the formation of a fistulous opening. When the obstruction occasioned by a stricture is very great, and it may be such that the passage will hardly admit a bristle, the urethra behind is dilated, often inflamed, and sometimes ulcerated, so as to give rise to urinary

fistula, or effusion of urine. The bladder and ureters are affected as we have before described. Of course the hypertrophy of the bladder, by propelling the urine more forcibly against the stricture, must tend to increase the dilatation of the canal behind. The urethra is sometimes obstructed by warty growths, which are situated generally near the meatus, and are remarkably vascular; they are developed as the result of gonorrhœa, and polypous growth is occasionally found, but is much more rare. Chronic diseases of the lacunæ sometimes converts them into small indurated tumors, which become imbedded in the corpus spongiosum; of this kind, perhaps, is an instance mentioned by Rokitansky, in which numerous cartilaginous protuberances, from the size of a millet seed to that of a pea, were scattered over the surface as far back as the bulb, not, however, obstructing the passage. It may be well to remind the student that the common expression of "old cartilaginous strictures" intend simply the density and firmness of the induration-matter, and not at all that it contains any true cartilage. Stricture is rare before puberty, but has been found at the age of ten years; it is well to be aware that it may at this age, possibly, be the result of the habit of masturbation. Mr. Adams enters his *caveat* against the "indiscriminate use of stimulating injections" as an occasional cause of stricture. Fibrinous exudation in very rare cases occurs primarily on the urethral mucous lining, and chiefly in children. It is probable that it takes place also here as a secondary process in the same diseases in which it is found in the bladder and ureters. Variolous pustules are not unfrequent in the urethra. *Tubercle* is of rare occurrence in the urethra; it is only present when the entire urinary apparatus is likewise affected. It has been found in the miliary as well as in the more massive form. *Cancer* affects the urethra, either in the male or female, for the most part, as an extension of adjacent disease; but cases are recorded where the growth in this part was either primary or isolated. Dr. Walshe is inclined to think that the vascular excrescence of the meatus in the female may "acquire a basis of scirrhus, or become infiltrated with encephaloid."

We notice separately the morbid conditions of the female urethra. Displacements of the uterus, especially retroversion, cause compression of the passage, as also does the pressure of the child's head during labor. Such compression not unfrequently produces sloughing of the parietes and vesico-vaginal fistula. *Dilatation* of the urethra is in rare cases congenital; it is sometimes effected purposely for the sake of removing calculi from the bladder, and may be safely carried to the extent of permitting a stone one inch and a half in diameter to be extracted. Paralysis, however has sometimes resulted, and consequent incontinence of urine from excessive dilatation. Prolapsus of the bladder alters the *direction* of the canal of the urethra so that it passes upwards and forwards. There is no essential difference between catarrhal inflammation of the female and of the male urethra; it is generally consecutive to a similar condition of the vagina. The lips of the meatus are seen to be swollen, and on pressure upwards muco-pus flows from the orifice. Stricture is very rarely, indeed, the result of inflammation, which appears to be owing chiefly to the circumstance that the disease

in the female is not of long duration. Mr. Curling met with a case in which a stricture, attended with complete retention of urine, was produced by contusion experienced during a severe labor. The *vascular tumor* of the meatus is thus described by Sir Charles Clarke: "Its texture is seldom firm; it is of a florid scarlet color, resembling arterial blood; and if violence is offered to it, blood of the same color is effused. It is exquisitely tender to the touch; and if an accurate examination of it be made, it appears to shoot from the inside of the urethra. Its attachment is so slight that it appears like a detached body lying upon the parts." Sometimes the growths extend partially along the urethra, or may even be situated at the neck of the bladder. The tissues of the urethra occasionally undergo a kind of chronic hypertrophy, so as to form "a bulbous tumor." The veins are enlarged and varicose, and the areolar tissue increased in quantity, while the mucous membrane may be either thick, or, on the contrary, thin and shining. A mucous discharge takes place from the canal and from the vagina. We are much inclined to consider this state as more truly deserving the name of chronic urethritis than that which Dr. Ashwell has so denominated, but which seems more of the nature of severe pruritus.

MORBID CONDITIONS OF THE URINE.

A brief *résumé* of these, including urinary concretions, seems properly to follow here. Urine is a fluid of amber color, of acid reaction, rather aromatic odor, quite clear and limpid, but depositing, after standing some time, a delicate cloudy sediment, varying in specific gravity from 1015 to 1022, and amounting in the twenty-four hours to from 30 to 40 ounces. Its quantity and its sp. gr., for the most part very inversely, so that in summer it is less abundant and heavier, in the winter more copious and lighter. The quantity of fluid taken of course influences the quantity of the secretion; if fresh spring-water be drank early in the morning, upon an empty stomach, it will pass off with extraordinary rapidity by the kidneys, so that the quantity of urine passed will be treble or quadruple the normal. Opium, in some persons, a few other substances, and certain emotions, have the effect of producing an unusually aqueous urine, as well as the unknown cause of the disease now called polydipsia.

The *acidity* of the urine varies very remarkably, according to the time which has elapsed since food was taken; it is less acid, or even alkaline, during digestion, especially of vegetable food, and becomes most acid when digestion has been finished for some hours. The urine is often alkaline from the presence of fixed alkali in healthy persons; it is important not to confound this condition, which will probably be replaced by acidity some hours after, with alkalescence from carbonate of ammonia, which is always indicative of disease. The permanence of the blue color of reddened test-paper, which has been dipped in the urine, will distinguish the former condition from the latter.

The *limpidity* of the urine is disturbed by a great variety of precipitates, which we shall presently notice. If the sp. gr. becomes very high,

the secretion at the same time not being diminished in quantity, it becomes an indication of disease, probably of diabetes. Diminution of the sp. gr., on the other hand, the quantity not being *temporarily* rendered excessive by any of the causes mentioned, is also a sign of disease; it may be of M. Brightii, or polydipsia.

The presence of *sugar constantly* in the urine constitutes the malady called diabetes, with whose true pathology we are as yet unacquainted; it seems, indeed, certain that in health the sugar in the blood is oxidized and converted into carbonic acid, while in diabetes the oxidizing process fails; but it does not appear that the proportion of carbonic acid expired is diminished in this disease, which certainly, according to the theory, should be the case. Sugar is detected with great readiness by the test called Trommer's, which consists in the reduction of the hydrated oxide of copper to the reddish-yellow sub-oxide, by the deoxidising agency of the sugar. Oxide of silver may be used as a test in the same way, and will be reduced to the metallic state if sugar be present. It must be remembered that uric acid and albumen will act somewhat in the same way as sugar, and probably other substances have a similar effect, but this is not so rapid or decided as that produced by sugar.[1]

Albumen is often present in the urine in its soluble form, and is best detected by the addition of a few drops of nitric acid, which render it insoluble, or coagulate it, so that it falls down as a whitish precipitate. It is necessary to remember, that the addition of acid may cause a precipitate of lithates, which is scarce distinguishable from that of albumen, except by being redissolved by heat; it is, therefore, always desirable to boil the fluid after it has given a precipitate with acid. Rare cases may occur in which the albumen, though present in considerable quantity, is so modified that it does not yield the ordinary reactions. Dr. Bence Jones has described a substance of this kind, which did not precipitate immediately by nitric acid, and when heated did not coagulate, nor was precipitated when nitric acid was added to the boiling urine.

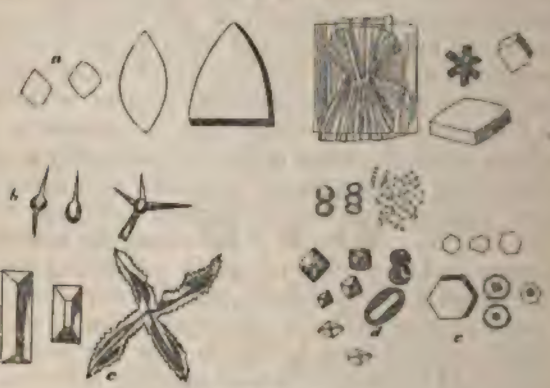
Chylous matter (so called) is occasionally present in the urine, but it appears that the essence of the disease does not consist in the flowing-off of chyle (from which the name is derived) constantly by the kidneys, but that, even during fasting, liquor sanguinis, with occasionally some trace of blood, escapes from the Malpighian tufts, and forms in the urine jelly-like coagula. In Dr. B. Jones's case, the influence of exercise in promoting and of perfect repose in preventing this unnatural drain from the blood was very marked, and showed clearly that it resulted from some peculiar defect in the organization of the Malpighian capillaries which rendered them unable to bear the increased stress of the somewhat excited circulation, without allowing more transudation to take place than was natural. The white color is due to the presence of oil in a finely-divided state, and is most abundant after food. Urine passed at this time forms sometimes a solid coagulum like blanc-mange, assuming the shape of the vessel in which it is contained.

[1] The only difference between ordinary diabetes (D. Mellitus, as it is termed), and the disease called D. insipidus, consists in the circumstance that the sugar, which is abundantly present in the urine, is tasteless. In composition, it is identical with sweet sugar.

The *coloring matter* of the urine is often of a much deeper tint than natural; this is chiefly the case in febrile disorders. In cachectic and exhausting diseases it often appears deficient, the urine appearing pale and wheyish. Urine of high color is often strongly acid, in moderate or diminished quantity, and tends to deposit uric acid crystals; it indicates, in some measure, a sthenic state of system. Urine, which is pale, is often alkaline or neutral, copious, more or less clouded by mucus or detached epithelium, and tends to deposit prismatic crystals of triple phosphate. This state is a faithful sign of asthenia. The color of the urine may be altered by hæmatin diffused through it; nitric acid and the microscope will then generally demonstrate the presence of albumen and blood-globules. Biliary pigment gives a remarkably deep tint to the urine, so that it is sometimes justly said to be like porter. This occurs, not only when there is actual jaundice, but generally when the liver is congested, and is a useful sign of this state. Its presence is demonstrated by the play of colors which takes place when nitric acid is added, the original tint passing through green, blue, violet, purple, to a pale red.

Urine is occasionally met with of a blue color, and this, from the presence of one of three different pigments, viz: cyanourine, indigo, and Prussian blue. It appears that these substances may be actually generated in the economy, though the latter two, of course, may be derived from without. Their pathological relations are quite unknown, and we must refer for an account of their chemical habitudes to Dr. G. Bird's work. Nearly the same may be said of melanourine and melanic acid, which are black pigments, giving the urine an inky aspect. They are, probably, peculiar modifications of hæmatin.

Fig. 264.



Urinary deposits.

(a) Various forms of uric acid.
(b) Urates, pointed, globular, and molecular (common).
(c) Triple phosphate, prismatic, and stellar forms.
(d) Oxalate of lime, octohedra, and dumb bells.
(e) Cystine.

A great many vegetable coloring matters affect the urine, and it should always be considered whether an abnormal tint of this fluid may

not depend on the presence of one of them. Logwood, beetroot, and rhubarb are, probably, the most likely to be met with.

The quantity of *urea* excreted varies according to the nature of the food taken and the amount of exercise. Lehmann, when living on an animal diet (thirty-two hen's eggs daily), found that his urine, in twenty-four hours, contained 819.2 grs.; when a mixed diet was taken, the quantity was 500.5 grs.; when it was purely vegetable, the quantity was 846.5 grs.; and when non-nitrogenous, it was only 237.1 grs. The quantity of uric acid varied similarly, though not in so great a degree. Exercise was found by Lehmann to increase the discharge of urea, lactic acid, phosphates, and sulphates; it diminished, however, that of uric acid, phosphates, and sulphates. Dr. Prout has noticed two morbid conditions, which are often termed *azoturia* and *anazoturia*, distinguished by an excess and deficiency of urea respectively. They are, probably, not so much essential maladies in themselves as symptomatic of certain disordered states of assimilation. Azoturia is detected by a formation of nitrate of urea taking place when nitric acid is added to the unconcentrated urine; it is not unfrequent in the oxalic acid diathesis. The quantity of urea in diabetes is considerably increased; in M. Brightii it is, as we have seen, greatly diminished.

Deposits of *uric acid* in crystals, or of its combinations with ammonia or soda, are exceedingly common. Now, with regard to these, it is first to be mentioned, that there are no certain evidences of an increased

Fig. 265.

Fig. 266.

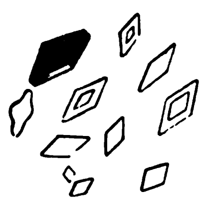


Fig. 267.

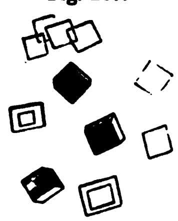


Fig. 268.

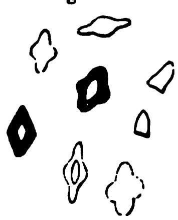
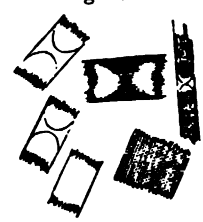


Fig. 269.

Fig. 270.



Uric acid crystals.

exertion taking place. Dr. Bence Jones has shown that urine, which appears thick from a deposit of urates, may contain actually less uric acid than urine which remains clear, in the proportion of 0.52 to 0.87. The appearance of uric acid crystals depends on increased acidity of the urine, though we do not certainly know what is the cause of this;

probably, however, the acid phosphate of soda. Three circumstances may occasion a deposit of lithates: (1.) An increased formation of them; (2.) Increased acidity of the urine; (3.) A low temperature. The primary form of uric or lithic acid is the rhombic prism; but there are innumerable modifications of it, chiefly depending on an elongation of

Fig. 271.

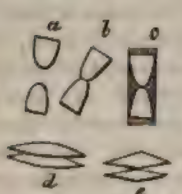


Fig. 272.

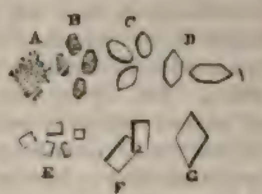
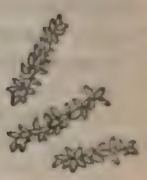


Fig. 273.

Fig. 274.

Fig. 275.

Fig. 276.



Uric acid crystals.

its long, and diminution of its short diameter, with rounding off of its angles, or on an approach to the rectangular form. The crystals often cohere in a radiated form. Their color is commonly a reddish yellow, derived from the urinary pigment, but they are, when pure, colorless. The common, so-called lateritious sediment, is in like manner colored by the pigment of the urine, and occurs sometimes nearly white. It consists of urate of soda, with small proportions of urate of ammonia and lime. This is positively stated by Lehmann, and we have satisfied ourselves of his being correct. Under the microscope, the deposit is seen to consist of minute granules, which cohere together in somewhat branching lines or shapeless masses. Occasionally, large opaque globules are seen, and separate, needle-like crystals; or the latter are observed radiating from a centre, which may be constituted by one of the large globules. These forms, however, we have scarcely observed, except in urates of potash, soda, or lime, artificially prepared. Their ready solubility by heat, and the deposition of uric acid crystals on the addition of acid, are sufficient tests for all urates. The appearance of a sediment of lithates can, in many cases, scarcely be considered morbid; it results from some diminution in the cutaneous transpiration, some

slight dyspepsia, &c. In febrile diseases, it is of very common occurrence, especially in gout and rheumatism. It is, however, particularly to be observed, that, in acute gout, before the paroxysm, and in chronic gout, the quantity of uric acid in the urine is diminished, while it accumulates in the blood. (Lehmann and Garrod.) In various diseases of the heart and lungs which interfere with respiration, in cirrhosis of the liver, at the close of paroxysms of ague, the urine deposits a sediment of lithates abundantly.

Phosphoric acid exists in the urine, combined with soda, ammonia, lime, and magnesia. The insoluble, earthy phosphates are held in solution by the acid phosphate of soda. When the urine becomes alkaline from the presence of ammonia, a deposit takes place, which, if the alkalescence be slight, consists of the monobasic phosphate of ammonia and magnesia $(HO, NH_4O, MgO)+P_2O_5$; if considerable, of the bibasic $(NH_4O, 2MgO)+P_2O_5$. The former is in the shape of separate prismatic crystals, the latter of dentated laminæ, radiating from a centre something like a star-fish. Simple star-like and penniform shapes of the monobasic salt are also described by Dr. G. Bird. Deposits of

Fig. 277.

Fig. 278.

Fig. 279.

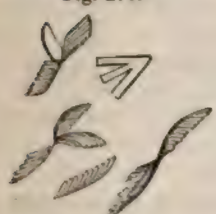


Fig. 280.

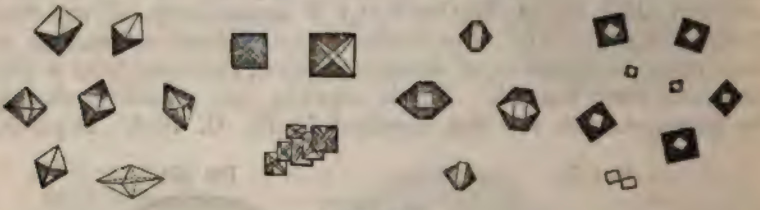
Earthy phosphates.

phosphate of lime are occasionally met with as opaque, amorphous, granular sediments. The presence of any of the phosphatic deposits generally indicates a feeble and depressed state of system, with nervous irritation. The quantity of earthy and alkaline phosphates in the urine may be greatly increased, without the appearance of any deposit, or any tendency to alkalescence. Dr. Bence Jones states that the alkaline condition, with its frequent accompaniments of triple phosphate crystals, "has no relation of any sort or kind" to an increase in the total amount of phosphates. The disease in which these are most abundant is acute

inflammation of the brain; in delirium tremens they are remarkably deficient, if no food is taken.

Oxalate of lime is not unfrequently found in the urine as a sediment which appears to the eye like a delicate mucous cloud, but is seen under the microscope to consist of multitudes of octohedral crystals, mingled in rare instances with dumb-bell forms. The pathological importance of a deposit of oxalates has been considered doubtful, and it is certain that it may easily be over-estimated. The conclusions which Mr. Coulson adopts (p. 79, of his work on *Diseases of the Bladder*) we believe to be correct. These are, that the occasional presence of a few crystals is no indication of disease; that the ingestion of aliments containing oxa-

Fig. 281. Fig. 282. Fig. 283. Fig. 284.



Oxalate of lime.

lates may occasion a deposit of this kind; that the same effect may be produced by sparkling wines or ales; that such crystals are often observed in the urine of those suffering from acute rheumatism, and in that of persons with emphysematous lungs, or who are short-breathed from other

Fig. 285. Fig. 286.

Dumb-bell crystals.

causes; that it is of very frequent occurrence at the commencement of convalescence from acute disease; but that its constant presence in the urine of persons who are not to be included in any of the above classes, is a valuable symptom of a peculiar dyspepsia, generally attended with a remarkable train of nervous symptoms.

Cystine, or cystic oxide, is of rather rare occurrence; it is said to be of scrofulous and decidedly hereditary character. A patient of our own, whose urine contained an abundance of it for some time, was very eccentric, and had a sister affected with mania. It forms a whitish sediment, consisting of hexagonal tablets of various size, mixed in our case with much fatty matter. The tablets are often serrated at the edges, and contain some oily matter in their centres.

Fig. 287.

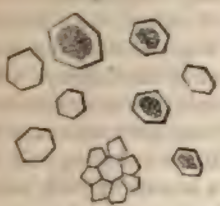


Fig. 288.

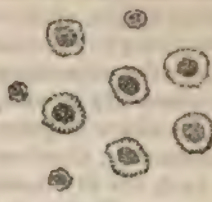


Fig. 289.

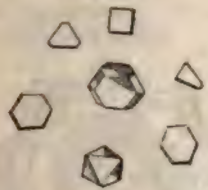
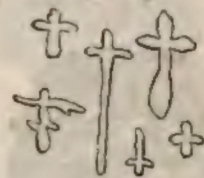


Fig. 290.



Cystine.

Carbonate of lime often occurs in the urine, in the form of an amorphous powder, when it deposits phosphates; it is produced by the decomposition of phosphate of lime by carbonate of ammonia, derived from the urea.

Calculi are concrete masses, made up of one or more of the various substances we have mentioned, the several particles of which, if crystalline, are held together by mutual attraction; if amorphous, are united

Fig. 291.

Lithic calculus.

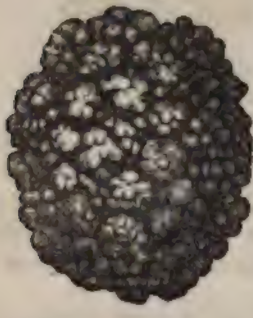
Fig. 292.

Section of a lithic calculus, showing the internal concentric layers.

together by some animal matter. Thus Dr. Walshe states the case, and no doubt correctly; but we have recently examined a small calculus, which consisted of octohedra of oxalate of lime, united together by a

considerable quantity of mucus, in which they were imbedded. The following description of the several varieties of calculi is an abbreviation of Dr. Prout's: (1) The *lithic acid* calculus is generally of a brownish-red, or fawn color, sometimes approaching that of mahogany. Its outer surface is commonly smooth. the sectional displays numerous concentric laminæ. Its shape is generally ovoid; its size very various; it is the commonest species; dissolves completely in liquor potassæ, and in nitric acid with heat, the dry residuum presenting a beautiful pink color. (2) *Lithate of ammonia* calculus is of a clay color, composed of concentric layers; its outer surface smooth, or slightly tuberculated; its sectional

Fig. 293.



Oxalic or mulberry calculus.

Fig. 294.

Internal structure of the same.

marked by concentric layers. It chiefly occurs in children under puberty, and hence is generally small, and rather rare. It behaves in several respects like the preceding, but is more soluble in water, and gives off ammonia when heated with caustic potash. (3) The *oxalate of lime* calculus is generally of a dark-brown color, from adhering and

Fig. 295.

Cystic calculus.

Fig. 296.

Internal structure of the same.

altered blood, but may be pure white. Its surface is rough and tuberculated (mulberry), its texture is hard and laminated. When heated it is decomposed, the acid being destroyed, and an alkaline ash (lime) remaining. Heated in a tube with sulphuric acid, carbonic acid and carbonic oxide gases are given off, and may be recognized by the former being absorbed by liquor potassæ, and the latter burning with a blue flame. (4) The *cystic oxide* calculus is of a yellowish-white; its surface is smooth, and of a crystalline aspect. It is not laminated, but appears

to be made up of a multitude of irregularly aggregated crystals, and has internally "the color and shining look of beeswax." It is soluble both in acid and alkalies, and crystallizes in hexagonal tablets from its ammoniacal solution. (5) The *phosphate of lime* calculus is of a pale-brown color, and smooth porcelaneous surface. It is regularly laminated, and the laminæ are vertically striated. It is not common, and does not

Fig. 297.

Phosphatic calculus.

Fig. 298.

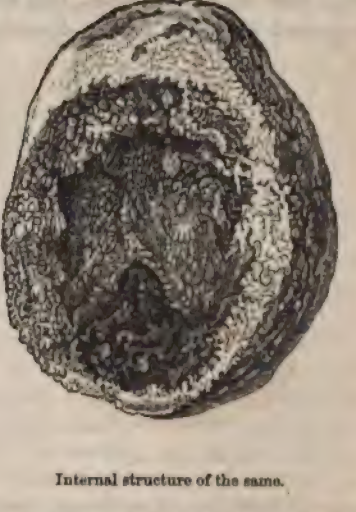
Ammonia magnesian calculus.

attain a large size; is soluble in hydrochloric acid, and precipitated from its solution by liquor ammoniæ as a white powder. (6) *Phosphate of ammonia and magnesia* calculus is nearly white, its surface is uneven; it is friable, and not laminated, except in some rare instances, when it

Fig. 299.

Fusible calculus.

Fig. 300.



Internal structure of the same.

is hard, crystalline, and more or less transparent and laminated. It yields ammonia when heated, fuses with difficulty, is soluble in dilute hydrochloric acid, and is precipitated from this solution by ammonia as prisms or stellæ. (7) The *fusible* calculus, a mixture of the two preceding, is whiter and more friable than any other, often of very large size, and occurs frequently. It melts readily, is soluble in dilute hydrochloric acid, and its ammoniacal precipitate consists of amorphous particles

and stellar crystals. (8) The *alternating* calculus is made up of two or more layers of different urinary deposits, as seen in the following examples; a nucleus of uric acid may exist with a covering of urates, oxalate of lime, or phosphates—the nucleus may be oxalate of lime with a covering of uric acid, urates, or phosphates; a nucleus of uric acid may be covered by oxalate of lime, and this by mixed phosphates, or the latter may be replaced by uric acid; again, a nucleus of oxalate of lime may be covered by uric acid, oxalate of lime, and phosphate of lime in succession. More than half the whole number of calculi are alternating, and it is especially to be observed, that, in a very great proportion of instances, the outer crust consists of phosphates; so that Dr. Prout has stated it as a law, that a decided deposition of the mixed phosphates is not followed by any other. (9) The *carbonate of lime* calculus is very uncommon; it is perfectly white, and very friable. We think it is occasionally found as a coating to renal calculi, being thrown out from the irritated mucous membrane. In one such case it appeared under the microscope as grains and round globules, about the size of those of the blood; it effervesces strongly with acid, and the lime after neutralization with ammonia can be precipitated by oxalate of ammonia. (10) *Uric* or *xanthic oxide* calculi are very rare; they are of a light-brown color externally, "and of a brownish flesh-color in their interior;" their surface is smooth and polished; they consist of concentric layers, and assume a waxy lustre on being rubbed. For its chemical characters we refer to Mr. Coulson's work, p. 282. (11) The *fibrinous* calculus is of small size, of amber color, and waxy consistence, and is probably only indurated fibrin, or mucus, therefore not a true urinary concretion.

CHAPTER XXXVI.

ABNORMAL CONDITIONS OF THE MALE GENERATIVE ORGANS.

Testicles and vasa deferentia.—There is no sufficient evidence to show that more than two testicles ever exist. They are absent when the entire sexual apparatus is wanting, and in some rare cases they are imperfectly formed, or one only may exist. An apparent absence of one or both glands at birth is not very unfrequent, the descent of the organ being arrested or delayed, so that it lies in the groin, the inguinal canal, or the lower part of the abdomen. Of one hundred and three male infants examined by Wrisberg at the time of birth, seventy-three had both testicles in the scrotum; while in twenty-one, one or both were in the groin, and the remainder had one or both in the abdomen. He found the imperfection more frequently on the left than on the right side, in the proportion of seven to six. Mr. Curling believes that if the descent does not take place within twelve months after birth, it is seldom fully and perfectly completed afterwards without being accompanied by hernia. The reason of this is sufficiently apparent, the pressure of the muscular walls of the abdomen must tend to cause the descent of the intestine through the open inguinal canal. When the testicle is still in the abdomen at birth, it may descend, and usually does, within a few weeks (it did so in ten out of the twelve cases mentioned by Wrisberg), or it may not descend till some time before puberty, or it may not appear at all. The cause of the testis remaining in the abdomen is considered by Mr. Curling, with much probability, to be owing either to paralysis and defective development of the cremaster muscle, or to the contraction of adhesions between the gland and some adjacent viscus. The discovery of the continuation of muscular fibres from the fixed attachment of the cremaster up along the gubernaculum to the testis in its primitive situation by the side of the vertebral column, inclines us strongly to believe that these fibres must be the agents in causing the descent of the gland into its appointed place. Contraction of the external abdominal ring is also mentioned as one of the causes impeding the descent of the testis. In rare instances, the testis wanders into other situations; one has been found in the perineum, the other being normally placed; and in two instances a testicle has preferred to make its exit by the crural instead of the inguinal canal. Sometimes it happens that the gland is turned round in the scrotum, so that its anterior face becomes posterior. It is quite ascertained that the abnormal situation of the testes in the abdomen is by no means inconsistent with the full discharge of their

function. Cases are occasionally met with in which the glands remaining in the abdomen have been small and undeveloped; but this imperfection could not be dependent on the unnatural position, as the analogy of animals and positive observation in men shows.

The vas deferens may be absent in a greater or less extent, and even the epididymis has been found in great part deficient. What is very remarkable is, that in several of these cases the testicle was fully formed, and though incapable of fulfilling its function, was scarcely less than the other. Experiments on animals have also shown that obliteration of their excretory ducts does not cause necessarily atrophy of the testes, nor efface in the individual the characteristic marks of the male sex. The most usual and least degree of this imperfection is that the vas deferens terminates in a blind extremity before reaching the vesicula seminalis. There is no such condition known as true *hypertrophy* of the testes; the gland may become greatly enlarged when attacked with inflammation, or when it is the seat of morbid growths; but while in the fullest activity of its function it does not exceed the normal size. This, no doubt, depends on its secretions, which of course may be formed in very varying quantities, rapidly passing of by the excretory duct. An increased production of semen by the testis would correspond to apparent hypertrophy of the thyroid by dilatation of its cavities.

Atrophy of the testis, either congenital from defective development, or acquired, is not unfrequent. Several instances are mentioned by Mr. Curling, in which the penis and testicles of persons arrived at the age of puberty, or of adults, did not exceed the size of those of children; two of these were of weak mind, but this condition is by no means the frequent accompaniment of cretinism or idiocy. A case recorded by Mr. Wilson shows the influence of aroused mental emotions in producing the due development of the generative organs, which had not taken place at the twenty-sixth year of age. The atrophy of the testis in old age comes on very gradually, the organ becomes flabby, and its tissues discolored, but is often little diminished in size.

Mr. Curling states that the ordinary weight of a sound testicle, in a healthy adult, is about six drachms, great individual differences, however, being often met with, as well as differences between the two glands; the left was heavier than the right in five cases out of six. If the weight fall below three drachms, the organ may be certainly said to be in a state of atrophy. "A testicle in an advanced state of wasting, not arising from disease of the gland, usually preserves its shape, but feels soft, having lost its elasticity and firmness. Its texture is pale, and exhibits few bloodvessels; the lobuli and septa dividing the lobes are indistinct, and the former cannot be so readily drawn out into shreds as before. The epididymis does not usually waste so soon, nor in the same degree, as the body of the testicle. It sometimes, however, loses its characteristic appearance; and I have even found it reduced to a few fibrous threads. The fluid pressed out of the wasted testicle and epididymis is entirely destitute of spermatic granules, and spermatozoa. In many instances, adipose tissue is deposited behind the tunica vaginalis, and encroaches on the epididymis and the posterior part of the testicle. Fatty matter is also found in the glandular substance of atrophied tes-

ticles. The structures composing the spermatic cord undergo a corresponding diminution, the cremaster muscle disappears, the nerves shrink, and the vessels are reduced in size and number. The vas deferens, though small, can generally be injected with mercury as far as the commencement of the epididymis. The testis of a man, aged thirty-four, dying of scrofulous disease of the lungs and kidneys, weighed little more than two drachms. The vas deferens and epididymis appeared healthy. The wall of the tubes had a fibroid aspect, and there were numerous fibre-forming nuclei on their surface. Their epithelial lining was everywhere in a state of fatty degeneration; it was reduced to atrophied, sometimes fattily degenerated, nuclei, and small corpuscles made up of oily molecules. The aspect of the gland tissue was dirty yellow; it was soft and flaccid. A testicle atrophied from disease is not only of diminished size and weight, but is altered in shape, being uneven and irregular, and sometimes of an elongated form. The surfaces of the tunica vaginalis are adherent, and its cavity is partly or entirely obliterated. There is no, or very little, trace of the proper glandular structure, the organ being converted into fibrous tissue of a firm texture. It loses its peculiar sensibility to pressure, but is sometimes the seat of morbid sensibility." The causes of atrophy of the testis are very various: deprivation of its supply of blood from obliteration of the spermatic artery, injuries of the spinal cord, producing paraplegia, and probably therewith the loss of the nervous influence necessary for the gland, inflammation of its tissue, over-excitement of the organ, the long-continued use of iodine, which is said to affect the female breast similarly, tubercular elephantiasis, injuries to the back of the head and nape of the neck, the pressure of effusions, and of large herniæ, neuralgia—all appear, on good evidence, to be real causes of atrophy of the part in question. The most common cause is inflammation; and it is easy to understand how the effusion of fibrin within the unyielding capsule of the gland should compress and obliterate the bloodvessels, as well as the tubuli seminiferi themselves.

Fig. 301.

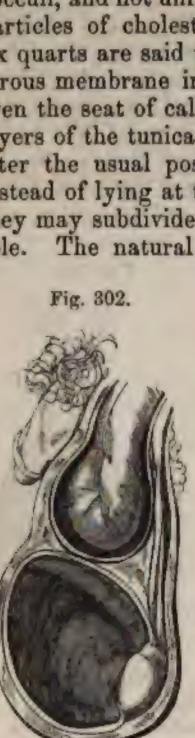
Inflammation of tunica vaginalis, after application of caustic; the aperture made by this is shown at*. There are flocculi of lymph on the serous surface of the testis.—From Mr. Curling's article.

The serous covering of the testis, the tunica vaginalis, is liable to be attacked by *acute inflammation*, and then suffers as other serous membranes do. The membrane becomes thickened and injected with blood, and is coated with a more or less considerable quantity of fibrinous exudation. Serum is at the same time effused into the cavity, and is rendered turbid by flakes of fibrin floating in it. The exudation may take the form of pus, but this rarely occurs. The unabsorbed fibrin very commonly forms adhesions between the opposing surfaces, which, in time, become very firm and dense, and may obliterate the cavity. It is by no means rare to find adhesions, to some extent, in the tunica vaginalis; Mr. Curling observed such in nine out of twenty-four in-

stances. The epididymis is prone to partake in the inflammation of the tunica vaginalis, and *vice versâ*.

Hydrocele is a dropsy of the serous covering of the testis, and does not differ essentially from dropsies of other serous sacs. The fluid is usually clear, and of a straw color, sometimes turbid, with albuminous flocculi, and not unfrequently contains a considerable quantity of shining particles of cholesterin. Its quantity is sometimes very considerable; six quarts are said to have been withdrawn in the case of Gibbon. The serous membrane in old hydroceles may be more or less thickened, and even the seat of calcareous deposit. Adhesions formed between the two layers of the tunica vaginalis may, according to their length and extent, alter the usual position of the testicle, so that it appears in front, instead of lying at the posterior and lower part of the distended sac; or they may subdivide the cavity, and produce thus a multilocular hydrocele. The natural cul-de-sac which exists between the epididymis and the body of the testicle, on the outer side, is sometimes much distended, so as to form a pouch, which projects on the inner side of the gland. The morbid action in hydrocele is confined to the serous membrane; the testis either remains natural, or is somewhat flattened, and, in some cases, partially atrophied by the pressure of the fluid. When, however, the original seat of disease is in the gland itself, the serous covering is often secondarily involved, so that serous effusion in the sac very often is associated with chronic orchitis, or other diseases of the testicle. This combination is termed a *hydro-sarcocele*. There is contradictory evidence on the question, whether hydrocele is more common on the right, or on the left. Mr. Curling's observations show that the right side is most often affected.

Fig. 302.



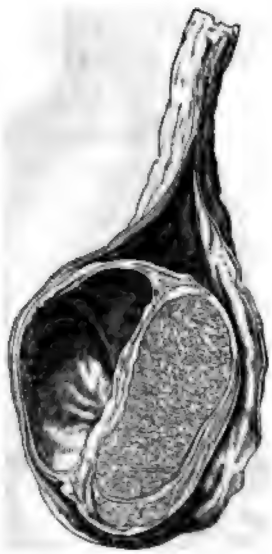
Drawing of large hydrocele, combined with scrotal hernia. —Curling's Art. on Testis, Cyclop. Anat. and Phys.

In *congenital hydrocele* the dropsical tunica vaginalis retains its fœtal communication with the peritoneal cavity.

Encysted hydrocele, as it is called, proceeds from the development of new cysts beneath the serous membrane. These are exactly similar to the simple cysts we have described, p. 183, having a wall of thin fibrous tissue, and a lining of tessellated epithelium, with usually limpid, fluid contents. They may be situated: (1) "beneath the visceral portion of the tunica vaginalis, investing the epididymis; (2) between the testicular portion of the tunica vaginalis and the tunica albuginea, which are thus separated from each other; (3) between the layers of the loose or reflected portion of the tunica vaginalis." In the two last-mentioned situations they rarely occur. When formed on the epididymis, they sometimes carry the serous membrane outwards as they enlarge, so that they become pedunculated, just as the small serous cysts in the neighborhood of the ovary often do with the peritoneum. Mr. Curling states that these pedunculated cysts do not acquire a large size, seldom exceeding that

of a currant. Several cysts may coexist in the same gland, and, when opened, produce the appearance of a sacculated arrangement. The walls of the cysts are liable to inflammation, which causes their contents to be mingled with various exudations of serum, fibrin, or even blood. Spermatozoa are very frequently present in the fluid of encysted hydroceles, to which they impart a milky or opaline opacity. They subside to the bottom of the vessel, where the fluid is left at rest, leaving the upper portion more transparent, but containing some albumen, which is not found in the limpid contents of ordinary cysts of this kind. It is extremely probable that they make their way into the cysts in consequence of rupture of some seminal canal lying in contact with them, just as a biliary duct sometimes opens into the sac of an hydatid. This opinion is confirmed by the circumstance that patients generally

Fig. 303.



Encysted hydrocele of tunica vaginalis.—From Mr. Curling's article. The cyst is between the tunica albuginea and the tunica vaginalis of the testis.

report the swelling to have commenced after some injury to the testicle. Spermatozoa are rarely, if ever, present in the fluid of common hydrocele. In one case, a few seemed to have escaped into the general cavity, from the rupture of a small cyst.

Diffused hydrocele of the spermatic cord is a rare affection. It consists in the enlargement of the cells of the areolar tissue, and their distension with a white or yellowish serous fluid. The inclosing fascial sheath is condensed and thickened, and at the lower part of the swelling, which is always the largest, separates it completely from the tunica vaginalis. The nature of the disease does not seem to be sufficiently ascertained. Mr. Curling classes it with simple œdema; we should be more inclined to regard it as a chronic inflammation.

Encysted hydrocele of the spermatic cord forms a tumor, of oval shape, and seldom attaining the size of a hen's egg. It is loosely attached to the vessels of the cord which pass behind it. Instead of there being only one cyst, there may be several, forming a series along the cord. This circumstance indicates their origin, from the partially obliterated process of peritoneum, which is carried down with the testicle in the formation of the tunica vaginalis. According to another view, they are simple cysts, of new production, analogous to those which constitute encysted hydrocele of the testis. It may be mentioned that simple hydrocele occurs not unfrequently together with some of the other varieties, and also with inguinal hernia.

Hæmatocele is the term applied to a tumor formed by an effusion of blood from the vessels of the testis, or of the spermatic cord. Its most

Fig. 304.

Hæmatocele: tunica vaginalis greatly thickened; testis pretty healthy.—From Mr. Curling's article.

common seat is in the tunica vaginalis, which may be so greatly distended as to exceed the size of the adult head. The blood undergoes various changes, coagula being formed, sometimes in separate masses, sometimes in firm layers, as in the walls of an aneurism. The fluid part, in old cases, is more or less thick and grumous, sometimes resembling coffee-grounds. The presence of the blood occasionally excites inflammation, leading to fibrinous and serous effusion, and, it may be, to suppuration. The effused blood sometimes putrefies, offensive gases are produced, and, unless free exit be given to the decomposed matter, fatal gangrene takes place. The tissues surrounding the tunica vaginalis are apt to become involved in the inflammation, though they are affected in a more chronic manner, and

thus the walls of the serous cavity are considerably thickened, perhaps so as to be half an inch in diameter. The testicle usually remains unaffected, except that in old cases it is atrophied from pressure. An encysted hydrocele of the testicle or epididymis may be converted into

Fig. 305.

Bruise of the scrotum, a form of hæmatocele.—Liston.

an hæmatocele, by the effusion of blood into its cavity. So, also, may the encysted hydrocele of the cord. Both these, however, are rare affections.

Diffused hæmatocele of the cord results from the rupture of some vessels of the cord, which are, probably, in some way diseased (*e. g.* varicose), in consequence of which blood is effused, in greater or less quantity, within the spermatic fascia; and, if the bleeding continues, or recurs after an arrest of varying length of time, a tumor of enormous size may be formed, reaching down even to the knees, as in a case recorded by Mr. Bowman, in the *Medico-Chirurgical Transactions*, vol. xxxiii. The cause of the rupture is generally some straining effort.

Orchitis (ορχις, a testicle), may be either acute or chronic. The acute disease is sometimes primary, as when the testis has suffered from external violence; more often it is consecutive, extending along the vas deferens, to the epididymis and testis. In the latter case, the disease has been called *epididymitis*, from this part being chiefly and most constantly affected. Opportunities rarely occur of examining the gland when acutely inflamed, but the following appearances have been observed: The testis itself is not much enlarged, owing to the unyielding nature of the tunica albuginea; its vessels are congested, so that it has a darker tint than natural. The epididymis is enlarged, especially at the lower part, to twice or three times its natural size, "and feels thick, firm, and indurated." This enlargement depends on the presence of exudation-matter. "The coats of the vas deferens are thickened, and the adjacent vessels injected. The tunica vaginalis is inflamed, and its cavity contains the usual effusions. Suppuration rarely occurs in the body of the testis in consecutive orchitis, it is more frequent in primary; indeed, in the former, it is not uncommon for the gland itself to escape entirely. When pus has been formed in the testis, it does not easily make its way

out, and consequently burrows in different directions, disorganizing the tissue of the gland. Sometimes, when all active inflammation has subsided, the fluid part of the pus becomes absorbed, and the remainder passes into the state of a concrete, whitish mass, much resembling crude tubercular matter. It may, however, be distinguished from this, by being contained in a cyst, and by the condition of the adjacent glandular tissue, which is more altered from its healthy state than it is in the neighborhood of tuberculous deposit. The color of the yellow exudation in chronic

Fig. 306.

Acute orchitis, attendant on gonorrhœa.

orchitis, and its relation to the tubuli, serve to distinguish it also from concrete pus. Another result of inflammation of the testis is wasting of the gland, occasioned by the interstitial fibrinous exudation passing into fibroid tissue, and compressing the bloodvessels and tubuli. The enlargement of the epididymis not unfrequently subsides very incompletely, leaving an indurated, knotty swelling, situated usually at its lower part. The fibrinous matter which imbeds the duct and the areolar tissue in this part, does not cause the obliteration of its canal, which is even sometimes considerably dilated, as Mr. Curling has observed, so as to be four or five times its usual dimensions. "In old cases, the epididymis acquires the density and consistence of cartilage, and sometimes even of bone." "The coats of the vas deferens are also found, for some extent, thickened and indurated."

Chronic orchitis is characterized by the effusion of a yellowish, homogeneous looking matter, in the substance of the testicle. This matter, when first formed, is soft, but afterwards becomes firm and solid, and intimately blended with the glandular tissue. It may occur as a single deposit, or as several coexisting in the same gland. These enlarge and coalesce, until they occupy the whole organ, giving it a uniform yellowish white appearance. In some cases, the epididymis is attacked by the same deposit, but, in the majority, it escapes. The seat of the yellow matter seems to be the interior of the tubuli. Mr. Curling, describ-

ing the condition of a specimen which he examined after injection of the bloodvessels, says, that at the anterior part of the testicle the deposit appeared as round, isolated bodies; "about the centre it assumed a beaded arrangement, and towards the mediastinum formed a number of closely-set yellow lines, or processes, radiating towards the posterior part of the testicle, where they were amalgamated into one uniform

Fig. 307.

Chronic orchitis, with fungous protrusion of testis.—From Mr. Curling's article.

mass." The deposit is usually considered, and we believe with justice, as of scrofulous nature, but Mr. Curling speaks confidently of its being coagulable lymph.

As the yellow deposit increases, it often causes adhesion of the two layers of the tunica vaginalis, as well as of these to the skin, and at the same time occasions ulcerative absorption of these layers, and of the tunica albuginea, so that, at last, a fungous protrusion of the affected tissue takes place. This projecting growth presents an ash or yellowish-white appearance, varied by irregular patches of a pale red hue, and sometimes of black. It is closely girt by the scrotum, the ulcerated edges of which are often thickened and everted. The fungous protrusion is not peculiar to chronic orchitis alone, a similar one may take place whenever inflammation of the gland has produced ulceration of the tunica albuginea, the softened and tumefied tissue within escaping at the opening, much as the brain does when hernia cerebri occurs from giving way of the dura mater. The result of chronic orchitis may be different to the above, but equally destructive of the efficiency of the gland. The inflammation, after it has given rise to a considerable amount of deposit, may subside, and the gland remain for a long time somewhat enlarged and indurated, but not painful. The deposit at last contracts and shrinks, inducing gradual atrophy of the testis, which may be reduced to a mere nodule, chiefly consisting of dense fibroid tissue, scarce larger than a nut. In these cases the epididymis often remains tolerably healthy, but is sometimes rendered "nodose, irregular, and hard."

Purulent deposits have been found in the testicle, apparently, as the result of pyæmia, in connection with synovitis of the shoulder-joint and lobular pneumonia. A preparation of this kind is in the Museum of St. George's Hospital.

Tubercle is not very infrequent in the testis; situated sometimes in

the body of the gland, but more often in the epididymis. In the former site, it appears as a single or as several masses of opaque yellow matter, which may increase, so as to occupy almost the entire gland—a thin layer of glandular tissue alone remaining around the morbid mass; on the tubercle softening and breaking down, a cavity may be formed, which ulcerates and discharges its contents externally through the scrotum. In the Museum of St. George's, there is a preparation which shows extremely well the cavity of a large scrofulous abscess of this kind, with a thick layer of tuberculous matter forming its wall. This is exactly analogous to a tuberculous cavity in the lungs. Mr. Curling gives a drawing of a specimen in which the epididymis was occupied by tubercular deposit in its whole extent, while the body of the testis remained perfectly sound. Cretaceous matter is occasionally found in the testis, which is doubtless the residuum of tuberculous deposit, which has softened and undergone calcareous change. It resembles exactly that which is found in the lungs and in the bronchial glands. Some uncertainty prevails respecting the actual site of tubercle in the testicle. Dr. Carswell regards it in this part, as elsewhere, to be formed on the free surface of mucous canals, or their continuations. Mr. Curling's conclusion we believe to be the more correct, that it may be deposited both interstitially, as well as within the tubuli. The tunica vaginalis is frequently inflamed when there is tuberculous deposit in the testicle. Pulmonary tubercle, or general tuberculosis, is present in many cases of similar disease of the testis.

Cancer is common in this part, and is most frequently primary. The *scirrhous* variety is occasionally met with, but is very rare; "it is characterized by its slow progress, as by its great induration." *Encephaloid* is the ordinary form of orchitic cancer; it commences as one or two masses among the tubuli, which it gradually destroys, as it accumulates. Whether it advances by infiltrating the tissue, as well as by pushing it aside, does not seem quite determined; the latter, however, seems to be the more common occurrence. As it increases in size it causes absorption of the tunica albuginea, which gives way, and allows the growth to sprout out, and to vegetate freely in the scrotum, which it distends sometimes to the dimensions of a fœtal head. The scrotum does not soon become involved in the disease, and ulcerated, probably in consequence of its distensile nature and loose connection with the testis. "The epididymis remains for some time unaffected." In one case, mentioned by Mr. Curling, its tubes were found filled with cancerous matter. Great enlargement of the vessels takes place; the spermatic artery has been observed as large as the radial. The spermatic cord becomes invaded by the disease, and sooner or later the lumbar glands, and perhaps the inguinal, become affected, while secondary cancers spring up in various parts. Encephaloid has been known to attack the testis in the first year of life, but is most common about the middle period. Its duration is very variable, from a few months to several years. *Colloid* cancer and *melanotic* have very rarely been observed in the testis. The tunica vaginalis is said to have been affected with fungoid (cancerous) disease, the testis remaining healthy.

Cyst-production sometimes takes place in the substance of the testis

in a very marked manner. The cysts may be only two or three in number, or excessively numerous, causing considerable enlargement. They vary in size, from the most minute to the dimensions of a pigeon's

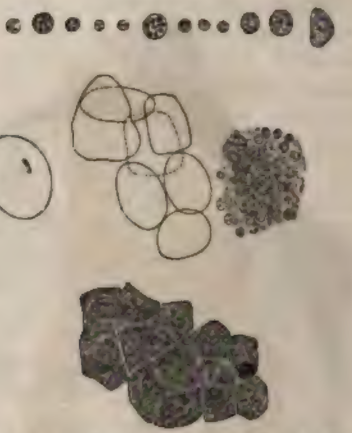
Fig. 808.

Section of cystic sarcoma of the testis.—After Mr. Curling.

egg. Their contents are, in the younger cysts, a transparent, light-colored fluid—in the older, a more thick, viscid, and very albuminous. The cysts are sometimes imbedded in solid stroma, probably of fibroid tissue; sometimes small masses of enchondroma are developed between them. A lobulated growth sometimes arises from the wall of the cyst, and occupies its cavity more or less completely. In one specimen we have examined, the contents of the cysts were not identical; in some, which appeared as opaque white spots, they consisted of layers of scaly epithelial particles, more or less flattened and pressed together; in others, the contents were a pulpy mass of swollen scales, with very abundant amorphous and oily matter, and small, delicate vesicular globules; others again contained a clear fluid, and a soft, whitish pulp, consisting almost entirely of small oil-laden, granulous vesicles. The stroma inclosing the cyst cavities consisted of a dense fibroid substance. Hemorrhage may take place into a number of the cysts at different places, as is well seen in a beautiful specimen of the disease in St. George's Museum. In this case, the testis was inclosed in a common covering—probably the tunica albuginea—along with the tumor, and lay at one side of it; in other cases, the glandular structure is expanded

over the growth. There was no contamination of the glands, nor, as far as known, any return of the disease in the patient from whom the tumor just mentioned was removed; but in other cases of cystic disease

Fig. 309.



Contents of various cysts in the case referred to in the text.
In the lowest figure, the epithelial scales are flat and opposed to each other; in the middle, the epithelial scales are much swollen, and mingled with granular matter and corpuscles.

the removal of the part has been followed by development of cancer in other localities. These latter are probably instances of the combination of cyst-production with cancer.

Loose bodies are occasionally found in the tunica vaginalis, similar to those occurring in joints. They are, in all probability, masses of exuded fibrin, and have, at least in the majority of cases, no claim to the epithet cartilaginous, which is often given to them on account of their external appearance. Calcareous matter is sometimes deposited in them; and Mr. Curling has observed the presence of laminæ similar to those of bone. The remains of a fœtus have been found in the scrotum, in connection with the testicle. This seems to have resulted from the inclusion of a second atrophied embryo in the abdomen of the first, from which it passed out with the testicle into the scrotum.

Morbid dilatation of the spermatic veins constitutes *varicocele*. In an advanced stage of the disease, the coats of the veins are thickened, so that they do not collapse when cut across. "The enlarged veins hang down below the testicle, and reach upwards into the inguinal canal; and, when very voluminous, conceal the gland, encroach on the septum, and extend to the other side of the scrotum." The veins in the interior of the testis itself, and those on the surface beneath the serous membrane, also become enlarged. The left veins are oftener affected than the right —partly in consequence of accumulation of harder fecal matter in the descending colon than in the ascending, partly on account of the left spermatic vein opening at right angles into the renal, and partly, perhaps, from the lower position of the left testis. When the varicocele is slight, it does not impair the nutrition of the testis; but, when large, it

occasions very marked atrophy—doubtless, in consequence of the increased venosity of the retarded blood.

Fatty tumors are occasionally developed in the spermatic cord.

It may be well to notice here the morbid conditions of the scrotum, as a portion of integument which has somewhat peculiar relations. It

Fig. 310.

Hypertrophy, or elephantiasis of the scrotum, in a Hindoo.

is one of the most frequent situations of *elephantiasis*—a disease which M. Cazenave regards as essentially connected with inflammation of the lymphatics of the part. This produces the most enormous enlargement of the part—such that the mass has been known to weigh 200 pounds more than the weight of the rest of the body. The epidermis, the corium, and the subcutaneous areolar tissue, are all, especially the latter, greatly hypertrophied. The areolar tissue is converted into a large mass of fibrous material, infiltrated with an albuminous and fibrinous fluid. Its areolæ are much enlarged in some parts; the testes remain sound; the spermatic cords are elongated several inches, owing to the testicles being dragged down, but are not otherwise diseased. Hydrocele sometimes occurs. When the disease is confined to the scrotum, and the enlargement becomes very great, "the penis becomes drawn in, and ultimately disappears, while the elongated prepuce is continuous at a navel-like opening in the skin of the surface of the tumor."

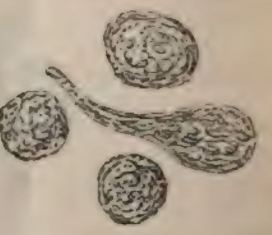
Common hypertrophy of the integument of the scrotum sometimes occurs; in this, there is no alteration of the subcutaneous tissue.

Epithelial cancer is the common form in which it appears in the scrotum, constituting what is commonly called *chimney-sweeper's cancer*, on account of its being apparently produced by the contact of soot. It is very remarkable that the disease may not appear for many years

Fig. 311.

An aggravated example of chimney-sweeper's cancer; much superficial texture destroyed.

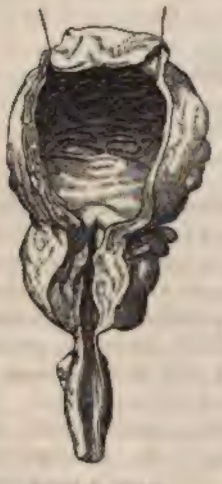
Fig. 312.



Corpuscles from chimney-sweep's cancer.

after the person has ceased entirely to be in any way exposed to the influence of soot. Mr. Curling mentions a case in which a man, after having been a sweep, went to sea, and led a sailor's life for nineteen

Fig. 313.



Enlarged prostate.

Fig. 314.

Hypertrophy of prostate.

years before the disease made its appearance. It is, of course, possible that, in these and similar cases, the cancerous development and the sooty employment were mere coincidences. Cancer of this kind does not show much tendency to contaminate the lymphatic glands or distant parts. Even the glands in the groin are not always affected, and those

in the interior of the abdomen very rarely. The disease advances by invading the adjacent tissues, and thus produces fearful ulceration, extending even to the groin and thigh, and destroying life by perforating the coats of some of the large vessels.

A case of *melanotic cancer* of the scrotum has been observed by Mr. Curling.

Fibrous tumors are sometimes developed in this part, and may form a large mass when several are grouped together.

ABNORMAL CONDITIONS OF THE VESICULÆ SEMINALES.

They participate in the defective development of the testes, being absent or imperfect when their related glands are so. Chronic catarrhal inflammation not uncommonly attacks the vesiculæ, causing tumefaction of their mucous membrane, secretion of unhealthy mucus, dilatation of the cavity, and thickening of its parietes. Ulcerative destruction of the mucous membrane occasionally takes place, the result of which may be thickening and cartilaginification of the parietes (the morbid action having subsided), or perforation, with formation of abscess in the adjacent parts.

Tubercular deposit occasionally affects the vesiculæ, chiefly in cases of extensive tuberculosis: it appears as "a thick yellow, cheesy, lardaceous, fissured, purulent layer," which replaces the mucous membrane, and causes thickening of the superficial layer of their coats. It never occurs before puberty. Scarce anything is known respecting *cancer* of these parts except that they only suffer secondarily from extension of adjacent disease.

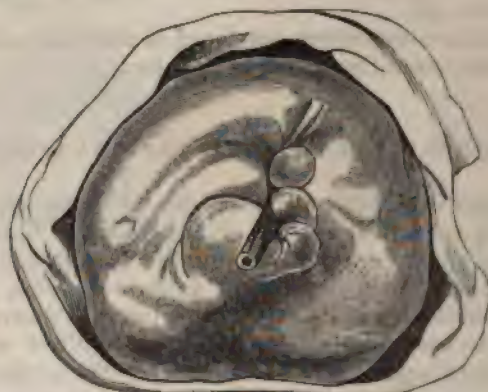
ABNORMAL CONDITIONS OF THE PROSTATE GLAND.

The prostate is imperfectly developed when the organs of generation are so. Its size is diminished in some cases, when the testes are atrophied; it is then rather consolidated in texture. In other cases it undergoes what has been termed eccentric atrophy; by which is meant the dilatation of its cavities, with thinning of its walls, in consequence "of the increase in size of calculous concretions in its follicles." "Cases sometimes occur, in which the whole of one lobe, or even the entire organ, is converted into a thin fibrous capsule, the proper substance of the gland being almost wasted."

Hypertrophy of the prostate is extremely common, and to some extent may be regarded as an occurrence natural in old age. It comes on quite imperceptibly, as one of those changes whose existence is unknown till their secondary effects begin to be produced. The enlargement may take place in all the dimensions of the gland, so that it expands laterally, downwards, and upwards. Most often, one lateral lobe is enlarged more than the other, which occasions a deviation of the urethra to the other side. It said that the left is the one most commonly affected, and that it also frequently projects more than the right towards the

cavity of the bladder. This, however, is by no means an invariable rule. The middle lobe is commonly enlarged more or less when the lateral lobes are so, but may attain a great size without any corresponding hy-

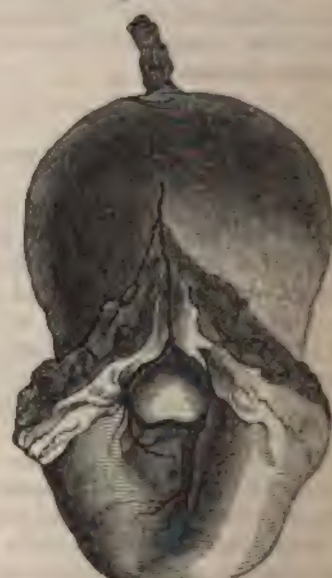
Fig. 315.



Lobulated hypertrophy of prostate.

pertrophy of the lateral. It forms a kind of pyramidal elevation, projecting into the cavity of the bladder, and causing the urethral orifice to

Fig. 316. Fig. 317.



Hypertrophy of middle lobe.

be raised, and in some measure blocked up. The enlarged middle lobe has been found of the size of a small orange; more often, it does not ex-

ceed that of a walnut; its surface may be smooth or nodulated. The hypertrophy of this part, when considerable, has the effect of throwing the neck of the bladder forward, and increasing the depth of its lower region so that calculi may lodge behind and below the prostate in its cavity. The canal of the urethra becomes lengthened in its prostatic

Fig. 818.

Irregular hypertrophy of third lobe.

portion; sometimes divided into two channels by the projection of the middle lobe; or is tortuous from being curved to one side; or, in consequence of its vesical orifice being raised, describes a curve whose convexity is downward. Its pressure on the rectum causes flattening of that channel, and more or less uneasiness in it, and may, perhaps, occasion hæmorrhoids, or prolapsus ani; in the same way it irritates the vesiculæ seminales, and induces thickening of their walls; pains felt in the parts to which the sacral nerves are distributed may be dependent on direct contact of these nerves with the enlarged gland, or on a reflected stimulus conveyed to their place of origin along the compressed vesical and hæmorrhoidal filaments; the most important result, however, of the enlargement, is the obstacle which it causes to the complete evacuation of the bladder. This depends partly on the narrowing of the urethra, and partly on the circumstance that a certain quantity of urine is always contained in the lower part of the bladder, below the elevated orifice of the urethra. In some cases the urethra, instead of being narrowed by compression, so as to appear like a slit on a transverse section, is considerably dilated, so that the prostatic sinus may contain two or three ounces of urine. An enlarged prostate is often indurated, so that by older writers it has been called scirrhous. The retained urine decomposing, causes irritation and inflammation of the bladder, with all its results, such as we have already described. Moreover, as Mr.

Coulson describes it, the enlarged middle lobe becoming broader, and raising up a transverse fold of mucous membrane, which passes off on each side to the lateral lobes, constitutes a kind of valve, which is pushed before the urine in every attempt that is made to void it, "and closes up the opening till the cavity of the bladder is very much distended; then the anterior part of the bladder being pushed forward, and the tumor being drawn back in consequence of the membrane of the posterior part of the bladder being put on the stretch, the valve is open, so that a certain quantity of water is allowed to escape, but the bladder is not completely emptied."

Acute inflammation may attack the prostate, commonly as the result of suppressed gonorrhœal discharge; it may go on to suppuration, or cause chronic enlargement, or an irritable condition of the gland, with increase of its secretion. When abscess occurs, there may be one or

Fig. 319.

Abscess of prostate.

several; a single one, large enough to contain half a pint of matter, or small and numerous foci, so that the gland appears riddled with holes. The abscess may open into the bladder, into the prostatic sinus of the urethra, into the rectum, or, externally, into the perineum.

Ulceration of the prostate scarcely occurs, except in some cases of chronic hypertrophy. It occasions severe suffering, and the admixture of blood with the urine.

Tubercle is not of frequent occurrence in the prostate; it is generally coexistent with tuberculosis of other parts of the generative apparatus and of the lungs. It may form a single large mass, or numerous small separate ones. As the tubercles soften and disintegrate, they give rise to abscesses, which pursue the same course as those of inflammatory origin.

Cancer is rare in the prostate; five cases of it only were met with by M. Tanchou among 8,289. The scirrhous species is scarce ever observed. Encephaloid, either primary or extending from the bladder, is almost the only form that occurs. It causes considerable enlargement of the gland, and may also perforate the mucous membrane of the bladder, and vegetate in its cavity, filling it up so completely as to give rise to the idea of the viscus being distended with urine. The disease occurred in a patient of Mr. Stafford's, at the age of five years, and in one of Mr. Solly's, at three.

Fibrous tumors, as Rokitansky and Mr. Adams testify, are frequently

found in the prostate. They vary in size from that of a pea to that of a nut; cause distinct hypertrophy of the gland, to whose tissue, however, they are, at least in some cases, but loosely attached.

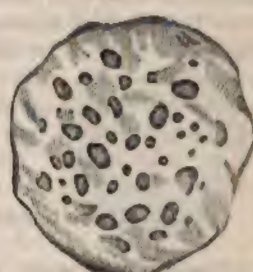
Fig. 320.

A cyst of the prostate gland.

Cysts are, in extremely rare cases, formed in the prostate, as Mr. Adams thinks, by closure of the outlets and dilatation of the cavities of follicles. The fold of mucous membrane constituting the uvula vesicle is sometimes so raised up by the increase of the subjacent tissue, or by that of the third lobe of the prostate, that it obstructs the free exit of urine; this is not to be confounded with simple enlargement of the middle lobe.

Concretions, in greater or less number, are of almost constant occurrence in the prostatic cavities; they often may be seen, on making a section of the gland, as reddish, yellow grains. The larger, fully formed ones have a beautiful laminated structure, and resemble a good deal a section of a lithic acid calculus. Their form varies very much; in the smaller it approaches the oval or circular, in the larger it is more polygonal or triangular. They are not unfrequently pale or colorless. They originate in large oval vesicles, formed of a well-marked homogeneous envelop. These appear to increase in size, while concentric laminæ are formed in their interior, whose interspaces are occupied by a finely-mottled deep-yellow or red matter. A central cavity is almost always left within the last-formed laminæ. Deposition of organic matter may take place, in some cases, exterior to the original envelop, but in most it appears to be within. Concretions of older date seem to lose the beautiful definiteness of their structure, and tend to disintegrate. The contents of these semi-organized formations appear to be earthy matter (phosphate, with a little carbonate of lime), tinged by the ordinary yellow pigment which is so often derived from the blood. We do not think they are developed from the ordinary epithelial particles of the gland, but that the original vesicles are cells of a particular kind, which are produced from organic exudation upon the mucous surface, and fill themselves, as their growth proceeds, with successive deposits of materials, which are probably poured out when the gland is the seat of vascular excitement. It is most probable that, in ordinarily healthy states, these concretions undergo solution at an early period of their existence, yielding up their contents to form part of the secretion of the gland. But, if this does not occur, and they go on increasing in size, they become the nuclei of, or are developed into, prostatic *calculi*. These are not unfrequently very numerous; as many as fifty or sixty have been found

Fig. 321.



Prostatic calculi.

in an atrophied dilated prostate, which has, in consequence, when examined *per rectum*, given the sensation of a bag of marbles. The calculi sometimes cohere together, and form a large mass, projecting into the membranous portion of the urethra, which becomes in consequence much dilated. A remarkable case of this kind has been recorded by Dr.

Fig. 322.

Prostatic concretions.

Herbert Barker,[1] in which 29 calculi, weighing together 1,681 grains, were cemented together so as to form a single concretion, which was nearly five inches long, and of an elongated pyriform shape. The surfaces of these calculi are faceted from mutual pressure; they are of a whitish or reddish color, of porcellaneous lustre and hardness, with a radiated, laminated, or compact structure. Lassaigne's analysis gives the following as their composition: basic-phosphate of lime 84.5, carbonate of lime 0.5, animal matter 15. The smaller calculi often escape into the bladder through the dilated prostatic ducts; if they remain there, they excite irritation of the mucous membrane and deposition of phosphates upon their own surface. A coating of lithic acid has sometimes been formed on a large calculus remaining in the prostatic portion of the urethra.

ABNORMAL CONDITIONS OF THE PENIS.

The penis is very imperfectly developed in some cases of normal condition of the other sexual organs, as well as when they are themselves imperfect. We have seen it extremely short and fissured in its whole upper surface, in a case of *eversio vesicæ*. When it is very small, fissured below, and destitute of prepuce, and when at the same time the testes remain in the abdomen, and the scrotum is cleft, there results a considerable resemblance to the female conformation; or if the penis is less atrophied, a pseudo-hermaphroditism. Atrophy of the penis, accompa-

[1] Transact. of Prov. Med. and Surg. Association, vol. iii. 1846.

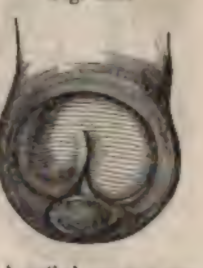
nied by obliteration of the cavernous textures, occurs, according to Rokitansky, together with atrophy of the testicles.

Hyperæmia of the penis, being in the exercise of its function a natural occurrence, scarcely ever seems to become morbid. It has, however, occasionally happened, during coition, that the erectile texture has given way in some part—probably from the rupture of some of the trabeculæ containing the small arteries. The consequence of this is, that the organ appears broke, and cannot assume the erect condition beyond the part injured. Contusions, which occasion bleeding from the urethra, indicating that laceration has occurred, are sometimes the cause of severe strictures; in some of these cases, inflammation may have been set up, resulting in the effusion of coagulable lymph; in others, the deposited fibrin of effused blood has furnished the induration-matter. "Inflammation of the cutaneous investment of the glans (*balanitis*), which is generally complicated with inflammation of the internal lamina of the foreskin, gives rise to excoriation, exudation of coagulable lymph, adhesion of the prepuce to the glans, suppuration, and ulceration; when chronic, it induces exuberant formation of epidermis; and if the deeper parts of the parenchyma of the glans are involved, obliteration, cartilaginous induration, and atrophy, follow."

The *vesicles of herpes* sometimes form on the prepuce, on its mucous or cutaneous laminæ. These are not to be confounded with the specific ulcerations, termed *chancres*, which may form on the internal surface of the prepuce, the frænum, and near the meatus within the urethra, as well as upon the glans, which is their usual site. We give Mr. Druitt's terse description of the appearance of the various forms of chancre, premising that he has selected the principal types:—

The *Hunterian* chancre is nearly circular, deep and excavated; the base and edges are hard as cartilage, but the hardness is circumscribed; there is little pain or inflammation; its color is livid or tawny. It may occur upon the common integument, the glans or body of the penis; in the latter situation it is never so hard and excavated as it is on the glans.

Fig. 323.



A vertical sore on a common site. The characters are chiefly those of the Hunterian chancre.—After Acton.

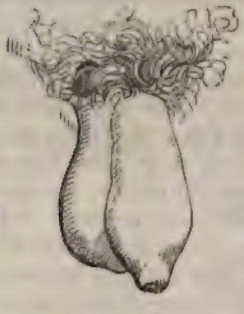
"The *non-indurated chancre* is more frequently found on the inner surface of the prepuce." It appears as a foul yellowish or tawny sore, attended with slight redness, and swelling and spreading circularly. It subsequently throws out indolent fungous granulations, unless it be situated on the glans, where they do not form.

Phagedenic chancres are of irregular shape, their edges ragged or undermined, their surface yellow and dotted with red streaks; their discharge is thin, profuse, and sanious. The surrounding margin of skin usually looks puffy and œdematous; but sometimes it is firm, and of a vivid red. The cicatrices left by chancres which have healed, are whitish, more or less hard, striated, and depressed.

Psoriasis of the prepuce produces a red, thickened, and fissured condi-

tion of the part, which bleeds, whenever an attempt is made to draw it back; and in consequence *phymosis* is apt to occur.

Fig. 324.



Phymosis.

Fig. 325.

Paraphymosis.

Paraphymosis is the opposite condition, in which a tight prepuce having been drawn back, constricts the neck of the glans, from having itself become thickened, and thus occasions a distended state of the glans, and even mortification, unless the stricture be removed. Phymosis is apt to give rise to attacks of balanitis, from the accumulation of the secretions of the coronal follicles.

Warty vegetations belonging to the class of epithelial tumors sometimes form on the glans, or on the inside of the prepuce; they are commonly the result of repeated inflammatory excitement, and are capable of being cured effectually by removal.

Fig. 326.

Warts on Penis.

Cancer may affect any part of the penis, but is most frequent on the glans and prepuce. Rokitansky says, that it chiefly assumes the encephaloid form; but we think the epithelial is more often met with, at least in the site which has been mentioned as its favorite. Dr. Walshe speaks of scirrhus as the species which usually affects the penis, though it may subsequently give rise to encephaloid vegetations. He states that the disease may originate as a warty excrescence, or as a pimple, which discharges an excoriating fluid, scabs, and breaks out afresh, while induration, followed by ulceration, advances at its base; or it may infiltrate the glans, so as to convert that part into an indurated mass; or venereal ulcers may take on cancerous action, and fungate as primary cancer. Secondary cancers, except in the adjacent glands, are not of common occurrence. Phymosis, and the irritation attending upon it, seem to act as exciting causes; advanced age as a predisposing.

THE PATHOLOGICAL ANATOMY OF THE FEMALE ORGANS OF GENERATION.

CHAPTER XXXVII.

THE EXTERNAL ORGANS OF GENERATION.

THE various tissues entering into the composition of the external organs of generation of the female are liable to numerous affections, differing according to the immediate seat of the lesion. In the pudenda, which we shall notice first, we have to deal with the cutaneous covering, the mucous lining, the loose, intervening cellular tissue, and the sebaceous and mucous follicles. The relation of the parts as the organs of copulation, is one, that, in addition to the pathological questions they give rise to, often has a most important bearing upon medico-legal points of vital interest, which it is necessary for the medical man to understand well, as numerous cases are on record of a misappreciation of the circumstances having led to very mischievous results. In the labia, sugillations are frequently met with as a result of external violence, or after parturition; the effusion from a violent injury may give rise to very considerable tumefaction, which must not be confounded with varicose swellings. When the consequence of childbirth, it generally affects the left labium,[1] and occurs more frequently in primiparæ than multiparæ. The swelling in either case has been known to attain the size of a fist, or a child's head. It presents a tense, smooth surface, with a livid color. Varicose veins of the labia may also acquire a very considerable size; but the slow increase of the tumor, and the vermicular character of its contents, will determine the diagnosis. Varicose swellings, too, occur during the course of pregnancy, and, though sometimes very considerable, do not generally cause any impediment to parturition, as they are external to the vulva. Cases, however, are recorded of their sudden laceration during parturition, and of a consequent fatal issue. The hemorrhagic tumor disappears spontaneously, or in consequence of treatment, but exceptionally the swelling persists, probably becoming encysted, and may then be borne for an indefinite period. A case is related by Mauriceau,[2] in which a tumor, originating in this way, existed for twenty-

[1] Kilian, die Geburtslehre, &c., vol. ii. p. 517. 1840. Frankfurt.
[2] Observations sur la Grossesse et l'Accouchement des Femmes. Paris, 1695. Obs. 29.

five years. Inflammatory affections of the labia may arise from internal and external causes, and exhibit the various forms of inflammation met with in other superficial textures. Eczematous and aphthous inflammation, as a result of derangement of the digestive organs, of pregnancy, of a want of cleanliness, or of sexual over-indulgence, are common. Eczema is characterized by the appearance of a vesicular eruption scattered over the inner or outer surface of the labia. The vesicles break and scab, and they are the source of much of the pruritus to which females are subject.

The loose cellular tissue, occupying the interval between the external and internal lamina, especially favors œdematous swelling, and, when the inflammation bears a phlegmonous character, extensive sloughs form. Instances of this in early life are recorded by Mr. Kinderwood,[1] who witnessed an epidemic at Manchester, marked by great fatality.

The mucous crypts, especially the aggregation lying on each side of the vestibulum, and termed by Bartholinus the female prostate, are liable to inflammation from catarrhal, herpetic, syphilitic, or other causes, resulting in chronic ulceration or tedious discharges. Even young children are frequently liable to simple or benignant inflammatory affections of these parts, giving rise to much irritation and muco-purulent secretion—a circumstance with which it is necessary to be acquainted, as popular prejudice is only too prone to attribute it to contagion.

The syphilitic taint gives rise to warty excrescences of the dermoid tissues, which may affect the labia and the introitus vaginæ. They consist of groups of small pedunculated tumors, aggregated together in such a manner as to produce a sort of mushroom appearance. These warts are not identical with, though they resemble, the tubercule muceux of French writers on syphilis. Mr. Safford Lee describes these as round, flattened tubercles, raised above the surrounding tissues, sometimes becoming elongated, of a reddish-blue color, and frequently ulcerated on their surface, producing a moisture of the parts.[2]

Encysted tumors of slow growth affect the labia, and are probably due to an obstruction in the first instance, and subsequent distension of one or more of the follicular structures. They consist of a membranous envelop, containing a transparent, glairy fluid; and only prove a source of inconvenience after they have attained a large size. Other tumors are described as occurring in the pudenda, independently of the hypertrophy resulting from chronic inflammation. Sir Charles Clarke has described a variety under the designation of the oozing tumor of the labium, which is chiefly characterized by a profuse watery discharge, corresponding in appearance with that from the cauliflower excrescence. It is but slightly elevated above the skin, and has an irregularly nodulated surface. It occurs in persons advanced in life, endowed with general obesity, and in whom the labia are enlarged. Erectile and scirrhoid tumors are also met with in this part of the system. A remarkable specimen of the latter is preserved in the Royal College of Surgeons of England (No. 2715), which was successfully removed by

[1] Medico-Chirurg. Trans. vol. vii. p. 84.
[2] Safford Lee on Tumors of the Uterus, &c. p. 254. London, 1847.

operation. It weighed upwards of eleven pounds, and is six inches in diameter. It is covered with healthy skin, and consists of a pale and compact, but soft and elastic, tissue, traversed in some parts by irregular, shining fibres, and in others having several small oval cavities in it. The patient was thirty years of age, and the tumor had been growing for many years. Under the head of hypertrophy, we must also allude to the liability of the labia being affected by elephantiasis. The nymphæ or labia minora are often abnormally enlarged, and frequently the seat of chronic inflammation, and consequent induration. In new-born infants they normally project beyond the labia majora, and, in some wild tribes, the custom exists of inducing their elongation by artificial means; this is said to be the case among the Bushmen and the Kamschatdales. Among the Arabs and Copts circumcision of females prevails, which consists in removing a portion of the elongated nymphæ. The enlargement of the nymphæ has been set down to an abuse of sexual indulgence; but this is, probably, as incorrect as the same statement has been shown to be with regard to hypertrophy of the clitoris. This rudimentary penis excites no attention, unless enlarged much beyond its normal proportions. It is capable of assuming the most extravagant size. Some of the cases of hermaphrodism that are on record may be explained by a reference to congenital hypertrophy of the clitoris. The largest specimen that we have met with is preserved in the Museum of the University of Bonn. It is fourteen inches in circumference, and weighs eight pounds. Mr. Safford Lee quotes several instances of similar hypertrophic enlargement. Parent Duchâtelet met with enlarged clitoris in only three cases of 6,000 registered prostitutes in Paris. Dr. Ashwell, in his remarks on the subject, expresses his concurrence with the last observer as to there being no necessary connection between an habitual sexual indulgence and the permanent increase of the clitoris. He adds, that he has often been struck with the integrity of the external genitals in prostitutes, while the uterus and ovaries have been bound in all directions by bands of false membrane. The warty growths, already spoken of, also affect the clitoris, and it is occasionally the seat of malignant degeneration, where the parts of generation are generally involved.

In the Pathological Society's Report for 1847–48, Mr. Brooke has recorded a case of malignant disease of the clitoris, which caused an excrescence of the size of a nut, attached by a pedicle, and which, having ulcerated and involved one of the nymphæ, was successfully removed by an operation. In this case it does not appear that the system at large was at all affected.

We allude to the urethra at present, only to speak of certain affections of the orifice which opens beneath the clitoris, into the vestibular portion of the vagina. The very large crypts and sebaceous follicles surrounding this sensitive point, are the frequent seat of blennorrhaic and other forms of inflammatory action. The mucous membrane of the part is liable to an hypertrophic development, giving rise to small vascular, generally pediculated tumors. They are exquisitely sensitive during life, and the surface being easily abraded by contact, they frequently exude small quantities of blood, or they are the cause of painful

micturition and protracted leucorrhœa. The mucous membrane surrounding the orifice of the urethra is very apt to become hypertrophied; the affection is described, by Sir C. M. Clarke, as consisting of an inflammatory hardening and thickening of the cellular structure, with an increase in the erectile tissue of the part.

The valvular fold of membrane which protects the virginal vagina, the hymen, which is commonly ruptured when coition is first completely effected, has been a subject of much discussion by medical jurists, as its absence has been regarded as an unequivocal sign of defloration, or its presence as a proof of the unimpaired virginity of the individual. Neither position is absolutely correct; for the best authorities are agreed, that, on the one hand, it may be destroyed by ulcerative absorption; or, on the other, that it may persist, not only after coition, but even after parturition. The latter fact is corroborated by the testimony of Merriman, Nägelé, Ramsbotham, and others. Other deviations from the normal state of the hymen are, the cribriform perforations that it exhibits; or it surrounds the entire introitus vaginæ, leaving a central circular orifice, or it entirely excludes the passage. The latter circumstance is not likely to be discovered, as other atresiæ of the external orifices are early in life. With the approach of puberty it will induce much inconvenience from the mechanical retention of the menstrual discharge, and, unless discovered and rectified, will be the source of serious disturbance. The hymen is sometimes found much indurated, and of a cartilaginous consistency, and even osseous deposits have been met with in it. The hymen, after it has been ruptured, is partially, if not entirely, absorbed. The carunculæ myrtiformes, which have been generally looked upon as the remains of the hymen, are now regarded by many authorities as normal formations that are not associated with lesion of the hymen.

THE VAGINA.

The vagina presents very considerable varieties of conformation and size within the normal limits of health, differences depending upon the age of the individual and the effects of cohabitation or childbirth, or the absence of these influences. A congenital closure of the passage may, independently of an imperforate hymen, or adhesion of the labia, convert the vagina into a cul-de-sac, a lesion which can scarcely be attributed to anything but intra-uterine inflammation, if the uterus be present. A remarkable instance, which appears to have been an arrest of development, is detailed by Dr. Boyd,[1] where, in a female, æt. 72, who had been married, though necessarily without issue, the vagina terminated in a cul-de-sac about half an inch deep, beneath the orifice of the urethra. There was no vestige of a uterus, nor any Fallopian tubes; the right ovary was natural, and attached by a loose ligament to the bladder; the left ovary was abnormal, but similarly connected with the bladder. A multiplication of parts is, perhaps, more frequently met with, and is produced by the formation of a septum, which

[1] Medico-Chir. Trans., vol. xxiv. p. 187.

is more or less complete; it may extend through the entire length of the vagina, or only partially divide it. A remarkable specimen of this malformation was exhibited by Mr. Birkett before the Pathological Society;[1] the vagina of a married woman, who had never borne children, and had died of pneumonia and pericarditis, was completely divided in the mesian line by a strong, dense, fibrous septum, extending from the external opening to the uterus; thus two vaginæ existed; each vagina led to a distinct os uteri, both of which were small; the neck of the uterus was rather longer than usual, the body smaller; the uterus itself was nearly divided into two cavities by a septum in the mesian line.

Occlusion, or stricture of the vagina, sometimes occurs as a result of external injury, or of cicatrization of ulcers. The rigidity or laxness of the walls varies much in different subjects, according to the general habit and the amount of secretion from the glandular apparatus surrounding the vagina. The great capability of the vagina for extension is best shown in parturition; hence, it is not to be wondered at that prolonged uterine or vesical disease should induce a very lax state of the mucous membrane of the vagina, which, as it often does, becomes a source of extreme distress and inconvenience to the party affected. In old women, we often meet with this relaxed condition, which may amount to a complete prolapsus. The anterior wall is particularly prone to be thus affected. Dr. Golding Bird has recently[2] pointed out that this lesion gives rise to a fetid, phosphatic, and mucous state of the urine in elderly females, owing to an accumulation of the urine in the prolapsed bladder lying in a pouch of the anterior vaginal wall. He shows that it may be the source of great irritability of the bladder and incontinence of the urine, which is best relieved by frequent catheterism, so as entirely to empty the bladder. Dr. G. Bird compares the condition with that resulting in men from enlarged prostate. In prolapsus of the uterus, the mucous membrane of the vagina is necessarily dragged down with the descent of that organ.

The vagina and the external organs are exposed to mechanical injuries of various kinds, and, in certain medico-legal questions, it requires care to determine their exact nature, as well as to avoid confounding the menstrual discharge with hemorrhage resulting from injury. Parturition frequently gives rise to laceration and severe contusions of these parts. The inferior portion of the canal, either from unusual rigidity, or from want of proper care on the part of the attendant, is apt to give way when the labor-pains are at their climax; and the lesion may vary from a mere laceration of the fourchette to a rupture of the entire perineum, from the vagina to the anus. It necessarily happens that the perineum is perforated before the infant reaches the natural outlet, and that it passes through the adventitious opening without establishing a communication with the former. Laceration of the upper portions of the vagina also occur to a varying extent, in conjunction with, or independently of, rupture of the uterus. A small laceration is not necessarily fatal. Ross[3] reports the case of a woman who was twice

[1] Report, &c., 1847-48, p. 295. [2] Medical Times and Gazette, January 1, 1853.

[3] Dr. Francis H. Ramsbotham gives this and other illustrative instances in his Principles and Practice of Obstetric Medicine and Surgery. London, 1841, p. 603.

the subject of an accident of the kind, and each time recovered. This result is out of the question, when, as occasionally happens, the child escapes into the peritoneal cavity.

The mucous membrane of the vagina is very frequently the seat of inflammation; the commonest form is the catarrhal. In the first stage, the passage is reddened, heated, and dry; this is followed by the secretion of a white, creamy mucus; or, if there be anything of a specific character, the discharge is more purulent or flaky. Whether simple or complicated, it often assumes a chronic character, and is then converted into a blennorrhœa, which, by the mere loss of fluid entailed upon the patient, often exerts a most debilitating effect. The secretion of the vagina in leucorrhœa has been recently shown by Dr. Tyler Smith,[1] to consist mainly of squamous epithelium and epithelial debris, though its essential characteristic, by which it is distinguished from the discharge derived from the cervix uteri, consists in its acid reaction, the interior of the cervix yielding an alkaline fluid. "It is to this alkali that the secretion within the cervix owes its viscidity and transparency, while the curdled appearance of the vaginal mucus is owing to the presence of the vaginal acid." As the acid of the vagina is sufficient to neutralize the alkaline secretion of the uterus, the fact of the latter being frequent and copious is masked; hence the discrepancy of the opinions of various authors on the subject of the source of leucorrhœa. The external surface of the os uteri, according to Dr. Tyler Smith, yields a secretion of the same character as the vagina itself. In both eczematous vesicles are frequently met with, which the same author regards as identical with the ovula Nabothi, which, by some have been interpreted as obstructed follicles, but Dr. Tyler Smith asserts that they are often found in situations where mucous follicles cannot be detected.

Rokitansky describes exudation, or croupy processes occurring in the vaginal mucous membrane primarily, but more frequently in conjunction with a similar disease of the uterus, in the shape of puerperal disease. "Exudative processes," he observes, "with various products, occur more frequently in patches, or throughout the vagina, as secondary diseases, both as a result of puerperal affection of the uterus, as well as in consequence of an infection of the blood proceeding from other causes, or from a degeneration of the typhous and various exanthematic processes. They correspond to the condition of the blood and its products, and accordingly produce a solution of the mucous membrane and the submucous layer, varying in shape and depth, and not unfrequently resembling gangrenous destruction. A loss of substance may ensue, and to this cause undoubtedly many cicatrices found in these parts are to be attributed." The secondary form of typhus occurring in the vagina, Rokitansky states, does not exhibit itself in the vagina in its genuine form, but is often found degenerated into croup and gangrene; an existing blennorrhœa, especially if of gonorrhœal or syphilitic origin, exerting a powerful attraction upon it.

A chronic thickening of the vaginal mucous membrane is occasionally met with; the follicular, the syphilitic, and the carcinomatous ulcer,

[1] Medico-Chirurgical Trans., vol. xxxv. p. 377.

also affect this part. Gangrene sometimes results from the effect of parturition, or the contusion caused by rough manipulation. The cicatrix that results from the healing of a slough is occasionally an impediment at subsequent deliveries; a puckering of the vaginal membrane, and consequent diminution of the passage, having taken place.

The vagina is not often the seat of morbid growths. Polypi and encysted tumors are the varieties that most frequently affect this situation. The posterior part of the vagina is stated to be the ordinary seat of polypoid growths.

An instance given by Mr. Curling, in the Reports of the Pathological Society,[1] forms an exception to the rule. Here the solid tumor which was removed from a woman, aged forty-five, grew from the upper part of the vagina, to which it was attached by a broad peduncle, which commenced just behind the meatus of the urethra, and extended backwards towards the uterus about two inches and a half. The structure of the polypi varies in character; they may be, as stated by Mr. S. Lee, fibrous-vesicular and cellulo-vascular—the fibrous being the least frequent. They vary equally in size, from a trifling projection to growths several pounds in weight. The encysted tumors of the vagina originate in an obstruction of the follicles with which the region abounds; they contain a glairy, transparent, greenish, or dirty-brown, albuminous fluid; and, though the source of irritation and inconvenience, are not productive of any danger: their correct diagnosis affords a speedy means of relief; but they have been repeatedly mistaken for totally different affections, such as prolapsus of the womb or the bladder, or for hernia.

Specimens of carcinoma affecting the vagina are preserved in most museums of pathological anatomy; they show that this part is commonly secondarily involved by an extension of the disease from the cervix uteri; "however, it may exist," to employ the words of Rokitansky, "though the latter is in a very undeveloped state, and even without it, in the shape of primary carcinoma of the vagina." The form in which it occurs is of the fibrous or encephaloid kind; malignant epithelial growths do not appear to affect the female organs of generation in the same manner as they occur in the male penis—a circumstance which may appear remarkable, as both possess a great analogy in regard to the component structures and the secretions they give rise to. The smegma præputii consists as essentially of epithelium as the vaginal discharges: and the perverted nutrition giving rise to epithelial cancer, may fairly be assumed to affect the secretory organs of the penis, and be regarded as an extravagant expression of the normal process. The fact, however, is, that the mucous membrane of the vagina has not been shown to be obnoxious to this form of cancer.

[1] Vol. i. p. 301, 1847–48.

CHAPTER XXXVIII.

THE INTERNAL ORGANS OF GENERATION.

THE UTERUS.

THE uterus, unlike most other organs combining to form the human body, has a double existence; one of long-continued comparative quiescence, and one of extreme, though brief, activity and development. The diseased processes affecting it are mainly associated with, and the result of the changes that take place in it, during and immediately after pregnancy; hence, its pathological relations scarcely come to the cognizance of the medical man until its proper functions have been called into action. Before considering its acquired abnormities, however, we must turn our attention to certain congenital anomalies presented by the organ, which have a bearing upon the future health of the adult individual. In a morphological and natural historical point of view, some of these malformations possess considerable interest; though we can only in so far advert to them as they regard the practitioner.

We have already given an instance (page 610,) of an entire absence of the uterus—a malformation which need not affect the health of the individual. Rokitansky states that the occurrence is extremely rare, and that most of the cases in which the uterus appears to be absent may be resolved into a partial arrest of development only, and that, by careful examination, we may find behind the bladder one or two rudimentary bodies in the proper fold of the peritoneum which represent the uterus. An actual multiplication of the organ is equally rare; but it is not an uncommon thing to find a more or less complete attempt at the formation of a double cavity, which is manifestly the result of an arrest of development. The bilocular and horned uterus are the malformations alluded to; in the former, a more or less perfect septum extends through the uterus in the mesian line; in the latter, the organ presents the character of the uterus exhibited by certain mammalia, as the sheep, and is divided into two lateral compartments by a fissure, extending vertically downwards from the fundus. This may be explained upon the assumption of an imperfect union of the two rudimentary bodies from which the normal uterus is developed. Only one of these may arrive at maturity, and we then have to deal with a uterus consisting only of a single horn, or of one-half; there will then necessarily only be a single Fallopian tube. The uterus unicornis, as well as the uterus bilocularis and bicornis, are capable of becoming impreg-

nated; Rokitansky[1] details the particulars of an example of pregnancy in a rudimentary uterine horn, which terminated fatally by rupture and sanguineous effusion into the peritoneal cavity, in the third month. When the two halves coalesce, the division which constitutes the malformation may vary considerably in amount; only a slight depression may be visible at the fundus in one case, so that the organ scarcely deviates from its normal condition; in another, the fissure extends so far down as to justify the appellation of double uterus, ordinarily bestowed upon the anomaly. An excellent instance of this is preserved in the Museum of St. George's Hospital (No. 104 of Dr. Lee's preparations). This preparation also illustrates what takes place after impregnation; while the ovum is received into one horn, which becomes duly developed with the growth of the fœtus, the other only sympathizes with it so far as to form a deciduous membrane, and thus to prevent the occurrence of superfœtation, but otherwise undergoes but trifling alteration or increase. Though impregnation and parturition are not necessarily fatal, these malformations seriously endanger the life of the patient—owing, as Rokitansky observes, partly to the want of the necessary dimensions of the part that undertakes the functions of the entire organ, partly to the obstacle opposed to the uniform development of the impregnated uterine half, by the unimpregnated half. These circumstances favor laceration of the uterine parietes. Rokitansky also shows that the divergence of the cornua from the axis of the body causes an impediment in the act of parturition, while the expulsive power of the uterus is much reduced by the absence, in the case of the uterus bicornis, of a true fundus.

Hypertrophy and atrophy of the uterus are, in part, normal at the periods of puberty and involution; much tact is necessary to distinguish some of the morbid from the healthy conditions of the organ. The weight and dimensions of the adult uterus fluctuate in health considerably. Kilian[2] gives the following, as the result of his measurements: the entire length varies from twenty-four to twenty-six lines; the greatest breadth is eighteen lines; the thickness, nine lines; the cervix is from ten to twelve lines long; its breadth, from six to eight; its thickness, from five to six lines; the length of the uterine cavity is twelve lines; its breadth, nine lines; the greatest thickness of the fundus, five lines; of the sides, four lines; and of the cervix, three lines. After one or more births, all these measurements increase from one-fifth to one quarter. The weight of the uterus varies from eight to twelve drachms, and may, after several pregnancies, amount to two ounces. With the aid of this table we shall be better able to determine whether we have to deal with a morbidly enlarged or diminished uterus. Either affection may involve the entire organ, or be manifested in a part only. An atrophic condition is probably a frequent source of sterility; the organ, and especially the cervix, is small and anæmic, its tissue dense, and the ovaries present an equally undeveloped condition. After the climacteric period, the cervix often disappears entirely, and nothing but an indurated ring remains at the sumit of the vagina.

[1] Pathological Anatomy, vol. ii. p. 277. Syd. Soc. Ed.

[2] Die Geburtslehre, &c., von Dr. H. F. Kilian, Frankfurt, 1839, vol. i. p. 92.

Hypertrophy is more commonly met with as a morbid state, than atrophy; partly, as an exagerated expression of the normal condition, at certain periods of life—partly, as the result of irritation, set up by other morbid processes. These may consist in tumors, occupying the substance, or filling the cavity of the uterus; giving rise, eventually, to actual expulsive efforts, resembling labor-pains, or to blennorrhaic affections of the mucous surfaces. Mere consensual irritation, proceeding from other organs, may suffice to induce it. Thus, the Museum of St. George's Hospital contains a preparation of the internal female organs of generation (x. 7), in which the ovaries are seen to contain cysts, while the uterus, which otherwise is perfectly healthy, exhibits very marked hypertrophy. The cervix is liable to be hypertrophied by itself; the labia may form a single tumefied ring, or present two tumors, lying parallel to one another, and separated by a transverse fissure. The first form is more likely to occur in women who have not borne children, and the second in those who have. The anterior is more frequently enlarged than the posterior lip.

The cavity of the uterus may be morbidly diminished in consequence of inflammatory affections of the surrounding textures, or by malposition or curvation, and may amount to complete obliteration. An instance of obliteration of the cavity of the uterus is preserved in the St. George's Hospital Museum (Dr. Lee's Preparations, No. 161), in which the cervix remained patulous. An instance of complete obliteration of the os uteri is recorded by Dr. A. T. Thomson, in the thirteenth volume of the *Medico-Chirurgical Transactions*, where, owing to this cause, in a female aged sixty-five, the uterus was found distended by eight quarts of brown fluid, slightly coagulated by heat. The patient had borne two children. We shall have occasion to see that the os and the cavity of the uterus are frequently plugged up by secretions, but this must not be confounded with actual adhesion of the parietes. Strictures are commonly met with at the external and internal orifices of the cervix; they appear to be mainly due to inflammation of the mucous and submucous tissues of the parts.

MALPOSITIONS OF THE UTERUS.

No organ of the body is liable to so frequent, and so varied and extensive changes of position, as the uterus; all of which very materially affect the health of the individual and her prospects of maternity. There are two great classes of malpositions; those in which the uterus maintains its site but alters its axis—deflections from the normal position—and those in which it quits its nidus, and becomes altogether displaced, so that its relation to all the pelvic viscera is perverted. The deflections are known by the terms anteversion and retroversion, in which respectively the fundus uteri is tilted forwards, or pushed backwards out of the ordinary axis. In either case, the abnormal position of the organ considerably interferes by its pressure with the functions of the adjoining organs, and especially the bladder and rectum, proportional to the amount of deviation. The term obliquity is applied to the lateral deviation from the axis which sometimes occurs, either as the

effect of pregnancy, or of diseased conditions affecting one side only, and thus disturbing the balance necessary to the integrity of the viscus. Much difference of opinion has existed in reference to the question whether anteversion (or, as it is called by some, pronation) is a more frequent occurrence, or retroversion. Lisfranc asserts that anteversion is by far more common than retroversion; Rokitansky positively states the latter to be the more ordinary occurrence. In these three forms of dislocation the different parts of the uterus maintain their proper mutual relations, but another variety exists in which the cervix and body form an angle (more or less acute) with one another. The deflection is almost invariably forwards; and may be congenital, as it is met with at early periods; and it is probably an impediment, though not a bar, to conception. Disordered menstruation accompanies most of the morbid conditions we have adverted to, but whether as a cause or as a complication has not been determined.

The second class of malpositions consists in a descent of the womb into the vagina, or in its extrusion beyond the labia; the term prolapsus has been, somewhat arbitrarily, applied to the lower degree, procidentia to the extreme form. In either case the axis of the womb must be altered, as well as its relations to the surrounding viscera. The predisposing cause is a lax state of the tissues generally, and more particularly of the ligaments of the uterus and of the vagina, which may be the symptom of debilitated constitution, as in lymphatic individuals, or the result of repeated pregnancies. The immediate cause is very frequently an unusual bodily effort. The secondary effect upon the prolapsed organ is, that it is irritated, and that its surface ulcerates, or that it becomes the seat of congestion and hypertrophy, and that its exposed surface becomes indurated and horny. Prolapsus is most frequently met with after the middle period of life; instances of its occurrence before puberty are recorded by Dr. Ashwell,[1] and other authors. Dr. Ashwell's work also contains the history of three cases in which, during the whole period of pregnancy, the womb had lain partly or entirely external to the pudenda. In two of these the child was born while the entire uterus was beyond the vulva; in one, it had occupied that position for several months, in the other for eight years previous to conception.

A very serious malposition, which comes on after parturition, spontaneously, or as the result of undue manual interference in removing the after-birth, in an unusually distended or relaxed womb, is that known as inversion of the uterus. It consists in a greater or less descent of the fundus uteri into the cavity of the organ, and it may amount to a complete turning inside out. It is generally accompanied by very dangerous hemorrhage: if the organ is not at once replaced in its proper position, and the patient survives the immediate shock, as sometimes happens, the uterus becomes reduced in size, and the inconvenience sustained may be comparatively trifling. Burns details a case in which an inverted uterus was borne for twenty years, menstruation continuing during the whole period.

[1] A Practical Treatise on the Diseases Peculiar to Women, p. 541. London, 1845.

Inversion is not, however, exclusively a sequel of parturition; it also occurs as a result of the influence of fibrous polypi, growing from the inner surface of the fundus. An unimpregnated inverted uterus is preserved in the Museum of the Royal College of Surgeons of England (No. 2,654), showing the Fallopian tubes obliquely in the upper part of the vagina—the effect of a polypus growing from the fundus. Velpeau removed a polypus from a woman, who died soon after of peritonitis, and the uterus was found to have been completely inverted. The presence of fibrous tumors in the substance of the uterus, or inclosed in the cavity, when complicating pregnancy, favors the occurrence of inversion, by disturbing the normal balance of the expulsive contractions. Some authors are inclined to attribute it to extreme shortness of the umbilical cord. Instances are recorded in which the entire inverted uterus has been removed by ligature, or by the knife; in some cases inadvertently, owing to the tumor having been mistaken for a polypus, and of the patient's having entirely recovered. One of the latest cases is that given by Dr. J. Cooke.[1]

We have already had occasion to allude to the occurrence of rupture of the uterus, as a concomitant of pregnancy in the horned or bilocular malformation of the organ. The accident is also met with in the normal uterus. A trifling laceration at the os tincæ occurs at every birth, and is, therefore, of no consequence; and it appears that until the solution of continuity extends beyond the circular fibres of the cervix, no danger is to be apprehended. Above this point the rupture may prostrate the entire thickness of the parietes, so as to allow an escape of the fœtus into the abdominal cavity; or one layer only, either on the inner or outer surface, may give way. It has been shown by several observers that the peritoneal investment of the uterus may, during parturition, alone be lacerated, leaving the uterine substance entire. The direction of the rent is stated differently by authors. Rokitansky affirms that it is generally vertical; Burns asserts it to be transverse, and Kilian maintains that it is commonly diagonal. It very rarely affects the fundus, but most frequently the posterior and inferior surface, which corresponds to the promontory, against which, in the act of parturition, the expulsive efforts propel the child with peculiar force.

Rupture of the uterus occasionally takes place before parturition, as a result of external injury; it is said not to be necessarily fatal, nor as dangerous as might be supposed. Its occurrence during parturition is unfortunately not a mere pathological curiosity. According to the statistics of the Dublin accoucheurs, Drs. Cullen and Clarke, the average frequency is about one in five or six hundred births. The former met with 34 cases in 16,414 births, the latter had 4 cases in 2,484 parturient females.

A remarkable circumstance is, that of the thirty-four cases that occurred in Dr. Collins's[2] practice twenty-three were male children, and he accounts for the fact by the circumstance that their heads are uni-

[1] On the Removal of the Uterus in Cases of Prolapsus and Inversion. London, 1836.
[2] Practical Treatise on Midwifery, &c. p. 244.

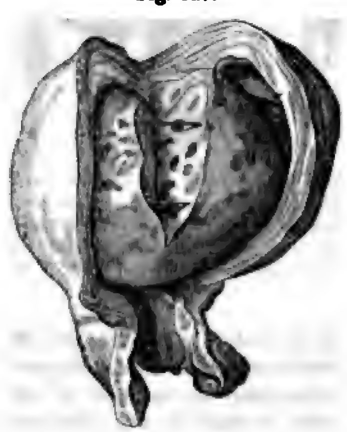
formly larger than those of female infants. The operation of turning is stated to give rise to the accident, and it also appears that primiparæ offer a greater liability than multiparæ.

MORBID GROWTHS.

In order to avoid unnecessary repetition, we shall postpone the consideration of the textural diseases of the uterus until after we have reviewed the morbid growths in the organ, in order that we may more conveniently connect the morbid states of the unimpregnated uterus with the diseased conditions occurring after parturition.

The abnormal formations that most frequently present themselves in the uterus, are fibroid tumors; they occur imbedded in the texture of the organ, or protruding from its minor surface into the cavity, or from some part of the external surface. When projecting into the cavity of

Fig. 327.



Fibrous tumor projecting into the cavity of the uterus.—St. George's Museum, 128.

the uterus they receive the name of fibrous polypi. While imbedded in the uterine tissue they form globular, white, glistening, dense tumors; there may be only one, or they may be numerous. In preparation No. 2,674, in the Royal College of Surgeons, we see a uterus, with from eight to nine large fibrous tumors in its walls, varying from one to four inches in diameter. In size they differ even more than in number; they are seen in every gradation, from that of a pin's head to that of a melon. The preparation spoken of further on is an instance of the great development they attain. Dr. Lee mentions one weighing fifty-four pounds, which contained several cysts filled with fluid. The fibrous tumor is surounded by a membrane which separates it from the uterine tissue, so that there is no very intimate union between the two structures. "The most usual position for these tumors," according to Mr. Lee's analysis of seventy-four cases, "is the submucous, viz: those projecting into the cavity of the womb, and the pedicles of these are generally situated

just below the openings of the Fallopian tubes. The next position in which they are most abundant is, the posterior wall and fundus of the uterus; they are very rarely situated in the anterior wall, and still more rarely in the cervix uteri." Of the general truthfulness of these remarks, every one may convince himself, by glancing through one of the metropolitan museums of pathological anatomy. It appears that the

Fig. 328.

A uterus, the upper half of which is enlarged by the growth of numerous fibrous tumors in its walls. One tumor, larger than the rest, projects into the dilated upper part of the cavity of the uterus, and completely fills it. Five others are shown by the section imbedded in the anterior wall, and many others project on the external surface of the uterus. The lower half of the uterus is healthy, but elongated. The walls of the portion occupied by the tumors are thick and laminated, like the walls of the uterus in pregnancy.—St. Bartholomew's Museum, xxxii. 16.

nearer the original deposit takes place to the mucous surface of the uterus, the more a gradual extension of the entire growth into its cavity is likely to ensue. In this way we account for the gradual elongation of the pedicle, which after a time is the only connection between the tumor and its matrix; it may then be removed with comparative facility by operative procedure. The pedicle is not, however, a necessary consequence of the arrival of the fibrous tumor at the external surfaces. In one of the largest specimens which has come under our notice, which is also remarkable as affecting the cervix (Royal College of Surgeons, No. 2,672) exclusively, the remainder of the uterus continuing normal, we find no attempt at the formation of a pedicle; the tumor has evidently formed in the substance of the posterior part of the cervix, and in its growth has separated the uterine tissue, which is still spread out over the upper part of the tumor, as if embracing it. The tumor, in this case, is twelve inches long, by five inches thick, and presents the ordinary structure of fibrous tumors. The fibrous tumors found almost free in the abdominal cavity, or, at least, only attached to the uterus by cellular adhesions, probably have the same origin as the growths we have just considered; having been developed in the first instance under the peritoneal investment of the uterus, they have subsequently become

detached. This does not preclude the possibility of their being formed primarily, at the points where they are found.

The intimate structure of fibroid tumors varies in some respects; to the naked eye it exhibits, at times, a concentric disposition of fibres; but more commonly, an irregular wavy appearance, without any uniformity of arrangement, presents itself; and it is more particularly in this case that cavities containing blood, a dark-colored gelatinous fluid, or a clear serum, are formed, which give the tumor, on section, a resemblance to the sero-cystic disease of the testis or mamma. Occasionally, the fibrous tumor presents a lobulated conformation. The microscopic appearances of the fibroid tumors of the uterus are not in accordance with what we should expect to find in a true fibrous structure. The microscope, in fact, demonstrates that they belong to an altogether different class of growths; the fibrous appearance is scarcely perceptible under the microscope, which displays elongated nuclei, imbedded in an amorphous stroma. It appears to us, that, from the analogy they present to the genuine uterine tissue, in the unimpregnated state, we should rather class them with homologous than heterologous productions; that they should be regarded rather in a relation to the womb analogous to that of exostosis to the matrix it springs from, than of a character totally at variance with that of their nidus.[1]

The amount of blood supplied to fibrous tumors, varies. The majority are but scantily provided with vessels. Some, when injected, only exhibit one or two larger vessels traversing the substance of the mass; others exhibit considerable and uniform capillary injection. The hemorrhage to which fibroid growths of the uterus may give rise, is not owing to a laceration of these vessels, but to the irritation and congestion they induce in the superincumbent mucous membrane, which, from the same cause, may ulcerate and slough. When complicating pregnancy, they induce hemorrhage, by preventing the normal development of the organ; hence, they are very apt to give rise to miscarriages. Fortunately for the individuals, they are often a cause of barrenness.

Not only the continued growth of these tumors, but also the occasional tendency to secondary changes occurring in them, manifest a greater vitality than some authors have ascribed to them. Thus, we find abscesses in the very centre of fibroid growths; or they may contain encysted melanotic tumors, as in the case of two preparations (Nos. 181 and 122) in the Museum of St. George's Hospital. A species of ossification or calcification occurs in these growths, analogous to the process of the same kind met with in other morbid products; the production of true cartilage, and the subsequent conversion into ossific matter, is at least doubtful, and not to be credited until we receive positive microscopic evidence to that effect. The calcification sometimes commences super-

[1] Since the above was written, a corroboration of the view expressed has been published in the Report of the Pathological Society for 1858, p. 219. Dr. Bristowe, in an elaborate paper on the subject of fibrous tumors of the uterus, concludes, from his examinations of them in the impregnated and unimpregnated conditions, that all so-called fibrous tumors of the uterus, at least in their earlier stages, before degeneration has taken place in them, are essentially muscular tumors; not simply fibrous tumors with a greater or less quantity of muscular fibre mixed up with them, but developments of true and undoubted muscular tissue.

ficially, at others, in the centre; the process seems to promote the spontaneous expulsion of the tumors from the uterus. When this occurs, the proper texture of the latter takes on similar action, as if a fœtus were contained in its cavity, and it becomes hypertrophied. This is not the case as long as the tumors occupy the tissue of the uterine parietes; here the pressure of the tumor rather inclines to produce atrophy; a remarkable example of this is spoken of by Dr. Lee,[1] in which, at least, in connection with a fibrous tumor at the fundus, the uterus had become so much atrophied as to resemble a mere bladder. Complete ossification may, however, take place, and the tumor be borne for an indefinite period, as in the case of the old lady mentioned by Mr. Arnott,[2] who, having died at the age of seventy-two, from the effects of a fall, was found to have a tumor weighing five pounds, and as hard as marble, in the uterine parietes, which had become converted into a mere membrane. The tumor had been diagnosed as scirrhus, when she was at the age of forty. It was found, on analysis, to contain nearly two-thirds of phosphate of lime.

Fibrous tumors have not been observed before puberty. Dr. Lee agrees with the statement made by Bayle, that they are most frequent in virgins, and that they exist in twenty out of a hundred middle-aged women.

POLYPI AND POLYPOID GROWTHS.

The growths which we shall next consider, though often confounded with fibroid formations under the name of polypi, are essentially distinct from them. These formations are soft and succulent, and project into the cavity of the uterus, or depend into the vagina; they are attached by a pedicle of greater or less width to the surface from which they spring, while they are invested by the mucous membrane of the part. They are essentially a morbid condition of the surface structures, the mucous membrane, the follicles, or sebaceous crypts of the different parts of the uterus. According to their predominant character, they have been termed by different authors—vesicular, mucous, cellulo-vascular, or channelled polypi, or polypi of the Nabothian glands. The last have nothing in common with an ordinary polypus; and, as Dr. Tyler Smith has shown, can only be regarded as a form of vesicular disease, affecting the cervix uteri. They are transparent cysts, one or more in number, and varying in size from a pin's head to a walnut, seated upon the cervix. They are generally sessile, but may become elongated, and thus acquire a pedicle. The mucous polypus is a pyriform projection from the interior of the uterine surface, identical in structure with the villi of the uterus, and hence, to be regarded as hypertrophy of this tissue. The cellulo-vascular polypus is described as a small red tumor, lying between the os uteri, and very much resembling the excrescences of the orifice of the urethra. These growths are very liable to give rise to hemorrhage, and especially at the menstrual period put on the character of erectile tumors; hence, they vary much in size, according to the quantity of blood they contain.

[1] Medico-Chirurgical Transactions, vol. xix. p. 94.
[2] *Ibid.*, vol. xxxiii. p. 199.

The vesicular polypus is stated by Dr. Lee always to be situated at the fundus, under the lining membrane, which is very thin and vascular; he describes it as made up of a number of little round vesicles or cells, which contain a thin, transparent fluid; the whole is supported by a thin fibrous tissue; they are of a dirty white color, and sometimes present a slightly yellow tinge. The channelled polypus of the cervix is a rare form of the disease; Dr. Oldham describes it as made up of several large channels, with occasional communications between them, and opening by large orifices on the free surface of the growth; it does not appear as a compound of pendent enlarged cysts, clustering together, but as a solid single polypus, with numerous orifices on its surface.

None of these soft growths can offer any impediment, as we see in the case of the fibroid tumors or polypi, to the act of parturition. When dormant, as they often are for a long period, they excite no symptoms; but they become dangerous when the seat of vascular excitement, by the hemorrhage to which they give rise. It is satisfactory to know, that the operation by ligature or excision, if properly performed, is a sure means of arresting it; a spontaneous cure has sometimes been effected by the constriction exerted upon the polypus, after it has passed the os uteri, by the circular fibres of the part.

CYSTS.

Cystic growths are extremely rare in the uterus. An instance of sero-cystic disease occurring here, is recorded in the first volume of the Pathological Society's Reports (p. 108). The tumor occupied the parietes of the organ, and presented a lobulated appearance; and some lobes seemed composed of separate bodies of various form, contained in and connected with the parietes of cysts. A portion of a uterus is preserved at the Museum of the Royal College of Surgeons of England (No. 2,657), in which, according to the catalogue, a very large encysted tumor had formed; the patient had been twice tapped and the cyst emptied; it was supposed during life to be ovarian dropsy. The occurrence of hydrometra, or a dropsical accumulation in the cavity of the uterus, is regarded by some authors as the result of a large hydatid forming in the latter; but it is probable that it is rather owing to the perverted action of the lining membrane pouring out fluid, which accumulates, in consequence of the occlusion of the os tincæ, and thus gradually distends the womb. Dr. Ashwell describes, under the same head,[1] the secretion and discharge of large quantities of limpid fluid, as a symptom of catarrh of the uterus; but it appears, from one of the cases appended to the chapter, that it may also accompany fungoid disease of the organ.

TUBERCLE.

Tubercular deposit in the uterus, affects primarily the lining membrane, where it is deposited in the miliary form, or accumulated in

[1] On the Diseases of Women, p. 506.

masses, aggregated into nodules, or forming a cheesy layer over the entire surface; the uterine tissue becomes secondarily affected, and is then liable to become infiltrated with the morbid product. When the affection has been accompanied by a discharge, Dr. Reynaud has shown that the vagina presents spots of ulceration, exhibiting a relation analogous to that of the trachea in pulmonary phthisis. The Fallopian tubes are generally affected coincidently with the uterus.

The rarity of the affection may be inferred from the fact that, among above two hundred phthisical females, Louis only met with three who furnished examples of tuberculous disease of the uterus. Considering how frequently the functions of the organ are changed, or arrested, during pulmonary phthisis, this is not the conclusion to which we should have been led by *à priori* reasoning.

CANCER.

Carcinoma of the uterus is a disease of frequent occurrence. Dr. Lever[1] has shown that the proportion of carcinoma to other uterine affections, is as one to seven, or about thirteen per cent. The period of life most obnoxious to it, is that between the fortieth and fiftieth years; and though numerous examples are met with earlier in life, the statement of Boivin and Duges, that in four hundred and nine cases they found twelve under twenty years of age, can scarcely be credited, unless, as we are assured by Dr. Walshe, uterine cancer is more prevalent in the French than in our own capital. The analysis of their cases yields the following table:—

Under 20 years of age	12 cases.
Between 20 and 30	83 "
" 30 " 40	102 "
" 40 " 50	201 "
" 50 " 71	11 "

They attribute a great share in the causation to sexual abuse, in which view they are perhaps strengthened by the comparatively large number of youthful victims. Messrs. Bayle and Cayol, however, emphatically deny that their researches lead them to a conclusion of a similar kind, but that they have found the disease occur with equal virulence in the lowest prostitutes, in married women, and in chaste girls. Dr. Walshe asserts that there is no shadow of proof that it ever owes its production to disproportionate or intemperate intercourse. Celibacy does not appear to favor its development; the ratio, according to Dr. Lever's analysis, is: single women, 5.83 per cent.; widows, 7.5 per cent.; and married females, 86.6 per cent. This is found to be identical with the relative frequency of other uterine affections in their respective classes. As a rule, the cervix is the part first affected; a feature which broadly distinguishes this disease from fibroid growths. A very remarkable exception is presented in a specimen of cancer of the body and fundus uteri in St. George's Hospital Museum, in which the cervix is entirely

[1] Medico-Chirurg. Trans. vol. xxii. p. 267.

free from disease (No. 134). The most eminent obstetric physicians are of opinion that many instances of so-called cancer are perfectly curable by proper remedial agents, from being nothing more than irregular thickening, and induration of the cervix, consequent upon chronic inflammatory action. It is, therefore, necessary to be careful in pronouncing an opinion in the earlier stages of the malady, and not to assume a patient to be affected with malignant disease, because of a more hardened and puckered condition of the os uteri. We possess no means of determining the nature of the affection during its first stage with certainty; nor is it frequently brought under the notice of the morbid anatomist. The advance of the deposit, however, and the concomitant subjective symptoms, the fusion and ulceration, the implication of the surrounding parts in the process, the fixation of the womb, and the rigid nodulated degeneration of the vaginal mucous membrane, soon enable us to form a positive opinion if the disease be malignant. Rokitansky has rarely found fibrous cancer affecting the uterus; this variety consists of dense, whitish, reticulated fibres, containing in their meshes a pale-yellowish, translucent substance; its limits are not sharply defined, but are lost in the uterine tissue. Rokitansky describes medullary carcinoma as the prevailing form of uterine cancer; appearing as an infiltration of a white lardaceo-cartilaginous, or loose encephaloïd matter, in which the uterine tissue is lost, and, like the former, giving rise to the nodulated surface of the conical portion of the organ generally regarded as characteristic of the disease. Colloid cancer, as Dr. Walshe observes, is probably never seen in the uterus.

The degeneration spreads more or less rapidly to the adjoining parts, to the vagina, the rectum, the other pelvic viscera and its osseous frame; and, in extreme cases, the whole contents of the abdomen are matted together, and present a frightful spectacle of disorganization and cancerous destruction. The ulceration that leads to this result, while it gives rise to fetid vaginal discharges, causes very extensive loss of substance of the parts first involved; the vaginal portion of the uterus, and the vagina itself, are the first to be eroded, and gradually communications are established between the various abdominal organs; the destructive character of the affection nowhere manifesting itself with the virulence that it here exhibits. Lebert, while admitting the dangerous character of the affection when attacking the cervix uteri, denies that it is genuine cancer; he views it as cancroïd, and therefore argues strongly in favor of local cauterization as a means of cure. He bases this opinion upon the frequent absence in cervical cancer of the genuine cancer-cell, and upon the rarity of the extension of the disease to the body of the womb. We have elsewhere developed our views with regard to the nature of cancerous disease generally; and, as we are unable to admit the existence of a specific and uniform cell-growth, characteristic of malignant affections, we must decline the inference of Lebert. He regards cancer of the fundus as undoubtedly deserving the name applied to it, both because the microscope detects the cancer-globule, and because the tissue of the organ is throughout degenerated. It is in the nodulated cancerous deposits, in this part of the organ, that he has often met with small purulent deposits.

Uterine cancer is ordinarily a primary affection; carcinoma of other organs may be developed simultaneously or consecutively; but, except as a result of the fusion of the former, and its consequent introduction into the system, it is not often the case. The average duration of the disease is stated by Dr. Lever to be twenty and a quarter months. The forms of uterine cancer, which we have spoken of, are not commonly liable to induce hemorrhage; in this respect, it differs materially from cauliflower excrescence of the cervix, a disease to which Dr. John Clarke[1] first drew attention. He describes it as an irregular projection, with a base as broad as any other part of it, attached to some part of the os uteri. The surface has a granulated feel, and is not tender; at this period the remainder of the cervix exhibits no sensible alteration, but by degrees the whole becomes involved. On removal from the body, it collapses, owing to its vesicular character. The rapidity of the growth varies; several observers differ from Clarke, in regarding it as of a malignant character; a view which is corroborated both by the revelations of the microscope and its power of reproduction after having been removed by the knife. Mr. S. Lee describes it as consisting of cells covered by an epithelial membrane; and, though he mentions the presence of compound cells, certainly concludes, from the absence of caudate cells, that it is a simple non-malignant structure. Both Rokitansky and Renaud regard it as a modification of encephaloid growth, accompanied by a remarkable development of capillary loops, closely resembling, as shown in the drawing accompanying Dr. Renaud's paper, the vascular arrangement of the fœtal placenta.

We may not conclude this subject without alluding to a fortunate accident in connection with "scirrhous growths" of the uterus, two instances of which are recorded by Dr. Ashwell, occurring in females respectively twenty-eight and twenty-one years of age. In both, large tumors broke up spontaneously, and were discharged per vaginam. Dr. Ashwell is clearly of opinion that they were cancerous; as such, they must have differed from the ordinary infiltrated character of uterine carcinoma, a circumstance that only adds to their peculiarity, the more so, as in each case the uterus appeared to have recovered its healthy condition.

THE VIRGIN UTERUS.

The textural diseases to which the virgin uterus is subject, are not of a character to occupy much attention on the part of the morbid anatomist. It is chiefly during the temporary physiological congestion, to which the organ is subject after maturity at the menstrual period, that morbid influences manifest themselves; and we then frequently have to deal with catarrhal and other slight forms of inflammation of the organ. Still, they often become of great importance to the individual, not so much from the intensity of the morbid process set up, as from the peculiar relation which the mucous lining of the womb, as a safety-valve to the entire organism, bears to the constitution of the female. The enact-

[1] Transactions of a Society for the Improvement of Medical and Surgical Knowledge, vol. iii. p. 321.

ments of the Mosaic ritual, and the prevailing customs of all civilized nations at the present day, equally acknowledge the necessity of attending to these indications. The extensive secretory apparatus, in the interior of the cervix, is chiefly liable to suffer, and to put on a chronic form of catarrh or blennorrhœa, marked by a viscid, straw-colored, transparent secretion, or by a more or less purulent and sanguinolent discharge. The rugæ, and deep intervening fossæ of the cervix, are apt to accumulate the secreted fluids, and the more adhesive they are the more a complete plugging up of the os uteri is likely to ensue; it is thus that catarrh may induce sterility; its extension to the lining mucous membrane of the uterine cavity further adds to this peculiarity, by impairing its functions, and rendering it incapable to prepare the proper nidus for the embryo. The same applies to catarrhal inflammation passing up the Fallopian tubes. It has been stated that the secretion of the cervix uteri is distinguished from that of the vagina, by the former being alkaline, while the latter is acid. It certainly is not so invariably, for in two cases, which we recently examined, the straw-colored mucus actually within the passage of the cervix was found to be strongly acid, while the microscope proved it to be made up exclusively of mucus-corpuscles arranged in strings. The effect of continued leucorrhœa of the cervix is to remove the epithelium covering the part, and to cause abrasion and ulceration. In the latter case, the villi are themselves destroyed; and this, as Dr. Tyler Smith has well shown, gives an eaten, corroded appearance to the mucous surface. Dr. Smith states that ulcers of the os uteri may be the primary result of inflammatory action, or arise from eruptive disorders of the mucous membrane, similar to herpes or eczema of the skin. But they more frequently result from the chronic irritation produced by the discharge from the cervix. This is confirmed by the fact, that, except in eruptive disease, the os uteri is rarely found abraded, unless there is coexistent disease of the glandular portion of the cervix. This circumstance, as well as the character of the majority of ulcers occurring at the part, have an important bearing upon our therapeutic proceedings. Taken in conjunction with the statistical records given by Dr. Lee,[1] they go a long way to disprove the necessity of the routine treatment of local cauterization, which has recently been insisted upon as an almost essential element in female therapeutics. The scrofulous diathesis favors both the profluvia of the cervix and deeper-seated destruction of its tissues; it gives rise to more profound ragged erosion on the surface and sides of the os, which, however, is not accompanied by that knotted induration which is generally characteristic of the carcinomatous ulcer. Nor is it marked by the same tendency to spread and involve adjoining parts in its destruction.

One of the effects of continued irritation of the uterine mucous membrane is hypertrophy, which induces a species of prolapsus, or the formation of polypoid growths at different points of the cavity, and occasionally stricture and occlusion, and consequent accumulation of fluid in the uterus.

[1] Medico-Chirurg. Trans., vol. xxxiii. p. 261. See also the admirable Lectures by Dr. West, very recently published, on Ulceration of the Os Uteri.

In addition to the forms of ulceration already spoken of, the uterus is subject to the specific ulcers of primary and secondary syphilis. Under the name of corroding ulcer, Dr. John Clarke has described a variety of malignant destruction which differs from genuine carcinoma only in not being accompanied by an indurated deposit. No account of the microscopic appearances of the part so affected is on record; a link is, therefore, wanting to enable us to pronounce positively as to the nature of the disease. It is of very rare occurrence; Dr. Ashwell, in the course of an extensive practice of twenty years, has only twice met with it.

Of the textural affections of the parenchyma of the uterus in the virgin state little need be said; a congestion of the organ appears to be a frequent source of malaise to the individual, and is recognized by the tumid, œdematous feel of the organ, and the injected purplish color of the parts when seen by the speculum; like other mere congestive affections, it rarely comes under the observation of the morbid anatomist.

CHAPTER XXXIX.

MORBID CONDITIONS FOLLOWING AND PRECEDING PARTURITION.

It is immediately after parturition that the uterus, which during pregnancy has become, as it were, the focus of the entire system, and, having completed the great cycle of its duties, is required to lapse into its previous dormant state—it is while the organ is yet the seat of increased vascular action, and its proper functions may be said to have ceased, that morbid influences are received with facility, and produce destructive and often rapidly fatal consequences. A large denuded surface is exposed to atmospheric contact; the process of absorption is rapidly going on, and any morbific matter, ponderable or imponderable, finds a ready recipient in parts in which normally the balance between health and disease is very even; add to this any depressing cause, acting on the susceptible mind or constitution of the female, and the balance is rapidly turned in favor of the latter. There are two states which are more particularly liable to supervene immediately after parturition, which are each of them a source of danger, by the hemorrhage they give rise to. The one is, atony or defective contraction; the other, spasm or irregular contraction of the uterus. In the one case, we find the uterus maintaining its dilated condition, its walls are flabby and soft; in the other, various irregular forms, to which the term hourglass contraction has been applied, present themselves. Both may become the subject of post-mortem examination, and instances are preserved in the museums of pathological anatomy. They are both allied to that influence which the vital powers sometimes sustain after parturition, from the severity of the shock to the system, and which, in its extreme form, may give rise to a fatal issue without producing any other symptoms but those of mere prostration. The various forms of inversion of the organ, which are in part due to a combination of these conditions, we have already alluded to.

PUERPERAL INFLAMMATIONS.

While the subject of fevers generally, and their proximate causes, are still under discussion, the nature of puerperal fever has been established with certainty to consist in inflammation of the uterus and its appendages; the various forms assumed by the febrile affections that follow parturition depend, in a great manner, as Dr. Lee observes, upon the serous, muscular, or venous tissues of the organ having become affected. Of forty

cases of puerperal fever, that had come under the notice of Dr. Lee, when he published the result of his researches, the peritoneum and uterine appendages were found inflamed in twenty-six; in fourteen, there was uterine phlebitis; in eight, inflammation and softening of the muscular tissue of the organ, and in four the absorbents were distended with pus. Almost the only exception among the classical writers on the subject is Dr. Copland, who asserts the occasional occurrence of a rapidly fatal form of puerperal fever in lying-in hospitals, in which there is no local lesion, but where there is a remarkable alteration of the blood, general lacerability of the tissues or loss of their vital cohesion soon after death, with a dirty, muddy, offensive, and sometimes serous effusion into the serous cavities. According to the predominant symptoms, nosologists have divided puerperal fevers into the inflammatory and typhoid, as Gooch and Boivin, and Dugès; into the inflammatory, the ataxic, and adynamic, as Tonnellé has done; or into the inflammatory, the synochoid, and adynamic or malignant, as proposed by Dr. Copland. Those who study the histories of the various epidemics of this fearful malady, will observe that, while there is a general resemblance between the local lesions, which more immediately concern us, the morbid symptoms during life vary according to the genius epidemicus, which is influenced by the predisposing as well as the exciting causes that are at work in each case. Here, however, there is also a general resemblance, which has been satisfactorily shown to present the same character as that of the erysipelatous constitution. That this consists in some poisonous agent capable of being communicated from one person to another, and analogous if not identical with the effluvia of putrefying animal matter, is rendered probable by the difficulty of arresting the spread of the disease when it has once appeared in a lying-in hospital; the strong evidence we possess of the communication of the infection from one person affected to another parturient female at a distance, through the intervention of the medical man, and by the direct effect of preventive means adopted with a view to destroy noxious effluvia and prevent contagion. Dr. Gordon, in his essay on puerperal fever, gives his adhesion to the view that erysipelas and puerperal fever possess strong analogies, and that they are concomitant epidemics. He observed the two together in Aberdeen, where they commenced together, kept pace together, arrived at their acme and ceased at the same time. Our limits forbid our accumulating evidence on this point; we may, however, add the testimony of one more observer in the Irish capital, Dr. Beatty, who, speaking of two epidemics that he witnessed, states that both attacks took place in January, and at each time erysipelas was raging as an epidemic in the surgical wards of the hospitals, and diseases of a typhoid type were very prevalent in the city. The strongest proof of the septic character of the disease has recently been afforded by the observations of Dr. Semelweiss, that, in the fatal puerperal fever which has long more than decimated the obstetric patients of the Vienna hospstal,[1] the poison was the cadaveric matter communicated by the students, who had previously been engaged in post-mortem

[1] See Dr. Routh's paper on the "Endemic Puerperal Fever of Vienna," Medico-Chir. Trans. vol. xxxii. p. 27.

examinations, to the females. The adoption of the proper precautions suggested by Dr. Semelweiss, as we have already shown in the chapter on phlebitis, at once reduced the mortality. Hence, it is manifest that one source of danger may be easily guarded against, and accoucheurs certainly cannot be too careful to cleanse themselves thoroughly after making post-mortem examinations, and handling putrid matter. Nor does it appear that this is enough, but that the medical man should, in these cases, be particularly on his guard to avoid every other possible source of contagion; "in the hour of the mother's peril," to use Dr. Holmes's eloquent words, "God forbid that any member of the profession to whom she trusts her life, doubly precious at that eventful period, should hazard it negligently, unadvisedly, or selfishly."

When the uterus has itself been the main seat of the puerperal inflammation, we find that an exudative process has given rise to the formation of a yellowish, or greenish, more or less gelatinoid lining on its internal surface, inducing a ragged, patchy appearance. This exudation may be easily detached from the subjacent mucous membrane, which according to the intensity of the disease is more or less reddened, tumefied, and softened. This condition may penetrate to the deeper tissues, and involve the entire thickness of the uterus, which will then, also, be more or less softened and discolored, infiltrated with a low sanious product, and even converted into a mere pulp. But we must guard against mistaking the dirty-colored, brownish, flocculent matter that is found investing the inner surface of the uterus soon after delivery, and which is merely the residue of the decidua, for the product of disease. This has been done by Boer and others, who have considered it as a proof of what has been termed putrescence of the organ. The rugged appearance of the part to which the placenta was attached also simulates this appearance. Dr. John Clarke states that this is only the remains of the maternal portion of the placenta, and of the coagula of blood left after its separation, which may be easily scraped off, showing the healthy surface underneath. He also testifies that he never met with mortification in any part of the uterus except in one instance, where it was manifestly attributable to the employment of instruments during labor. Rokitansky describes putrescence as the lowest form of uterine inflammation, and states that it differs from ordinary sphacelus; in this case, the internal layer of the organ is covered with a thin, opaque, or more dense product, varying in color from pale green to dark brown, beneath which the tissue, to a greater or less depth, is converted into a similar pulp. Small abscesses are sometimes found in the muscular tissue, without any perceptible change in the surrounding parts; generally, however, the structure of the muscular fibre is entirely destroyed. Tonnellé, who has analyzed two hundred and twenty-two cases of puerperal fever, in which he performed post-mortem examinations, found the womb affected with simple metritis in seventy-nine; in twenty-nine there was superficial, in twenty deep softening.

The more the inflammation presents a sthenic character, the more the products will resemble those of healthy inflammatory action; and, conversely, the more ataxic or adynamic the disease, the further they will be removed from that type. Rokitansky considers metritis to be

essentially of a croupy character, the peculiar nature of which, he says, is fixed by the form of the product, the condition of the subjacent tissues, and especially by the state of fusion exhibited by them.

METROPHLEBITIS.

Of the tissues entering into the constitution of the uterus in pregnancy, and immediately after parturition, none appear to possess a greater proclivity to be attacked with inflammation than the venous channels and the lymphatics; both may be primarily and coincidently affected, which is generally the case, or they may be attacked separately and secondarily. According to the observations of Tonnellé, who found that, of two hundred and twenty-two fatal cases of puerperal fever, one hundred and thirty-two presented inflammation of the veins and lymphatics of the uterus, phlebitis alternates with inflammation of the muscular tissue; during the great epidemic which this author followed in 1829, the latter occurred very frequently at the commencement of the year, and disappeared in July and August, and recurred in the months of September and October. The intervening summer months were characterized by the frequency of phlebitis.

Uterine phlebitis is generally set up within the first twenty-four hours after delivery, and though it cannot be invariably traced to the orifices of the veins, where the placenta adhered, it is probable that the introduction by them, of foul exhalations or secretions into the vessels, is the main cause, both of the local and the general effect. In the more favorable cases, adhesive inflammation prevents the spread of the malady; the danger is increased if pus forms, and the most virulent symptoms result if the product is of a sanious or septic character. Dr. Lee is of opinion that it not unfrequently occurs without proving fatal, and he bases his supposition upon the fact of calcareous concretions and disorganizations of various kinds being frequently met with in the spermatic and hypogastric veins of aged females, which he attributes to previous phlebitis.

In uterine phlebitis we find the organ studded with small abscesses, which may be traced to the vessels; these present the various appearances described under phlebitis, modified according to the character or duration of the disease in the individual case. When the lymphatics are affected, they, like the veins, become varicose, and thickened and distended with the purulent or sanious products of inflammation.

Uterine phlebitis is very commonly followed by metastatic abscesses; it is to the occurrence of the secondary purulent deposits, and the coincident infection of the entire system, that the main source of danger may be attributed. The more adynamic the type of the disease, the more probable it is that pus will be absorbed, and the local circumscription of the disease prevented.

PHLEGMASIA DOLENS.

As a disease immediately secondary to uterine phlebitis, we must now turn to the consideration of Phlegmasia alba dolens; Dr. Davis deserves the credit of having first shown it to consist in inflammation of the crural vein; Dr. Lee first succeeded in tracing the uterine origin of this affection anatomically; he demonstrated the inflammation commencing in the branches of the hypogastric vein, and subsequently extending from them to the iliac and femoral trunks of the affected side. The cellular tissue surrounding the vein participates in the inflammation, and that, as well as the impeded return of the venous blood to the heart, gives rise to much œdema of the limb, and condensation of all the tissues. This may be followed by suppuration, or sloughing; it may terminate in a complete cure, by resolution; or, in a partial recovery, with obliteration of a portion of the vein, and permanent induration of some of the soft parts. In a case examined twenty-one months after the attack, Dr. Lee found the external iliac vein, with its subdivisions, and the upper part of the femoral, converted into a ligamentous cord, so that it could only be distinguished from the surrounding cellular tissue by careful dissection. No traces of the entrance of the common iliac into the cava could be made out. The left side has a greater tendency to become affected than the right. The pathology of phlegmasia dolens is considered, by Dr. Copland,[1] of a more complete nature than we have described it; while he does not deny that it most frequently consists in inflammation of the femoral and iliac veins, he thinks it occasionally commences in the nerves, and sometimes in the lymphatics and glands, the veins then being secondarily affected. Nor does Dr. Copland admit that it is always referable, for its origin, to the uterus; he attributes the lesions in the veins, in some instances, to the consequences of prolonged pressure, or to this cause, and the sudden removal of that pressure, the disease originating in the iliac and femoral veins; again, he is of opinion that it may originate in lesion of any of the pelvic viscera, or of the parietes of the pelvis, or that it may be unconnected with any affection of this character, and owe its origin to rheumatic influences directly acting upon the lower extremity.

PUERPERAL PERITONITIS.

The serous membrane of the abdomen is very frequently attacked by inflammation in the puerperal state. Peritonitis occurs primarily or secondarily, but the form in which it presents itself, as well as its existence, depend much upon the character of the epidemic; thus, while M. Tonnellé found evidence of peritoneal inflammation in 197 out of 222 cases, Dr. Bartsch, in Vienna, under whom 109 fatal cases of puerperal fever occurred in the fourteen months preceding December 31st, 1834, found that it seldom exhibited the form of peritonitis, but gene-

[1] A Dictionary of Practical Medicine, vol. iii. p. 254.

rally appeared as metrophlebitis. If we examine the table of puerperal epidemics, collated by Dr. Churchill,[1] we find that, while of forty-three, in which the predominant post-mortem appearances are given, only four were characterized by uterine phlebitis, twenty-nine are stated as peritonitis. It is then, undoubtedly, the lesion most commonly associated with puerperal fever. It may be limited to the surface of the organ, and more particularly to the part surrounding the neck, or it may involve more or less of the entire sac. In two epidemics, it appears that the omentum was the seat of election.

Dr. Joseph Clarke states that the surface of the stomach, the liver, the spleen, omentum, great and small intestines, uterus, and the internal peritoneal lining of the muscles of the abdomen, will, in their turns, or altogether, be found to partake of the disease; but, as far as his experience goes, no part more than another.

The membrane never exhibits much vascularity, and in the low typhoid forms there is a remarkable absence of congestion and redness. In the more sthenic forms, which approach to the character of ordinary peritonitis, the greater vascular action is accompanied by the production of lymph and pus of a healthy appearance, adhering to the surfaces and matting them together. The ordinary character of the exudation, however, is a copious effusion of an aplastic character; the abdomen then contains from a few ounces to several quarts of serum of a dirty-yellow, greenish, or brownish hue, in which flocculent particles of lymph are floating, while but small patches of a thin non-coherent exudation are observed on the peritoneal sac. A remark of Dr. Hodgkin,[2] in reference to the small quantity of tender false membranes often met with accompanying serous effusion into the peritoneum applies here; it is to the effect that, though unquestionably of inflammatory origin, they are, in many cases, to be regarded as the result of a secondary action which the presence of the fluid has exerted in the peritoneum.

The smell of the fluid is also distinctive; it will be recognized when once noticed, as it differs from anything met with in the human body, in health or disease. The fluid is described by the older authors as of a creamy character; hence, the long prevailing fallacy that it was connected with an actual metastasis of milk, which was in a measure supported by the failing supply of the mammary secretion observed as one of the first symptoms of the disease. The serum contains a comparatively small portion of albumen, offers an acid reaction, and is said to possess a very salt taste. Beyond an analysis by Dr. Pearson, which is not equal to the requirements of modern chemistry, we have not met with a minute determination of its constituents. The ovaries and Fallopian tubes are very frequently found inflamed in puerperal inflammations; neither, however, are ever primarily affected, but coincidently with, or consecutively to, the diseased conditions just considered.

[1] Essays on the Puerperal Fever, &c., by Fleetwood Churchill, M. D. (Sydenham Society), p. 31.

[2] Lectures on the Serous Membranes, &c., p. 150.

COMPLICATIONS OF PUERPERAL FEVER.

We conclude this subject by a brief glance at the morbid processes accompanying puerperal fever in other organs, in which we shall closely adhere to Rokitansky. The entire track of the intestinal mucous membrane is slightly reddened and invested by a secretion of a thin serous or viscid gelatinous or more or less purulent character; the mucous membrane fuses, and the submucous tissues are infiltrated. To this circumstance we attribute the diarrhœas accompanying puerperal fever. The mucous membrane of the colon occasionally presents a dysenteric exudation, resembling that found on the internal surface of the uterus. Similar processes are also met with on other mucous surfaces, as of the respiratory, urinary, or œsophageal tracts. Dr. Clarke, in alluding to the occurrence of aphthæ in the mouth and fauces, as a frequent symptom in puerperal fever, and to their also being found at the anus, denies the correctness of the statement that the aphthæ go through the whole intestinal canal, as it is not borne out by dissection. The serous and synovial membranes of the whole system sympathize with the morbid processes of puerperal fever; the pleura are almost constantly found to contain exudations similar to those met with in the peritoneum; the pericardium contains them less frequently; the articulations very commonly exhibit exudations of a fibrinous or purulent character. The dura mater often presents a slight reddening, with a thin, soft exudation. To the secondary abscesses, resulting from capillary phlebitis by the extension of the inflammation of the uterine veins, or by the absorption of pus, we have already alluded. Rokitansky describes a black softening of the mucous lining of the fundus ventriculi, or of the œsophagus, indicated during life by black vomit, as a frequent occurrence. The stomach may become ruptured, and the black matter be thus effused into the abdominal cavity.

The blood exhibits various changes; its fibrinous coagula present a viscid, greenish-white appearance, or the coagula are scanty, gelatinous, and soft. The blood is of a dirty brown red, or chocolate color, and glutinous, or it is much attenuated and transudes all the tissues. The fibrin may form vegetations on the valves from mere mechanical deposition. The jaundice often affecting women during the puerperal state, is not dependent upon an appreciable lesion of the liver, but upon pyæmia.

DISEASES OF PREGNANCY.

The consideration of the morbid processes complicating parturition is appropriately followed by an account of the diseased conditions met with in the placenta and in the ovum; we shall, at the same time, touch upon extra-uterine pregnancy.

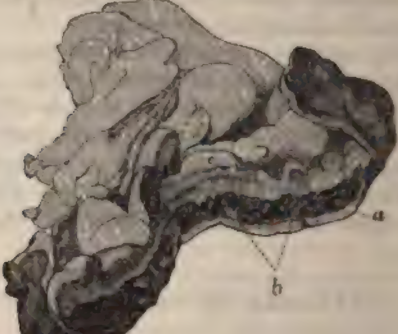
THE PLACENTA.

The placenta varies much in size within the limits of health. Its position differs also considerably without inducing any detriment to mother or child; but when placed near or over the os uteri, the frequent hemorrhages that occur endanger the lives of both. The umbilical cord, instead of being attached to the centre of the placenta, is sometimes inserted at the edge; this gives rise to what has been termed the battledore placenta, a deviation which, though not in itself perilous, may become so by rough manipulation after the birth of the child. The same applies to those cases in which the vessels of the cord are divided before they reach the placenta, or are inserted into the membranes. Other irregularities of the cord consist in its being excessively short, or extravagantly long, and in its being tied into knots. The extremes of length on record are two inches and fifty-seven inches.

Concussion, or other external violence, is a frequent cause of partial separation of the placenta, inducing extravasation into the tissue, and frequently giving rise to abortion. That the placenta is the seat of numerous morbid processes, may be inferred from the close relation it bears to the nutrition of the fœtus, the frequency of fœtal disease, and the necessary transition through the placenta of any morbific agent, which induces the latter. It is only, however, very recently that the attention of pathologists has been directed to the diseased conditions of this organ; our knowledge of the morbid changes to which it is liable is, therefore, as yet, very limited.

Professor Simpson[1] describes congestion of the placenta as affecting

Fig. 329.



Sectional view of atrophied placenta. The atrophy and fatty degeneration of the maternal and fœtal portions were caused by a fibrinous layer on the uterine surface. *a.* Fibrinous deposit. *b.* Maternal portion of placenta. *c.* Fœtal portion.

the maternal or fœtal portion, causing the external surface of the organ to assume a more or less deep violet, and, sometimes, almost livid color,

[1] Edinburgh Medical and Surgical Journal, vol. xv. p. 265.

the internal structure presenting a deep-purple hue, from the vessels being overcharged with blood, while the substance is heavier and more solid than natural. One of the sequelæ of congestion is hemorrhage into the body, or on the surface of the placenta, varying much in extent. The effused blood undergoes the changes usually traced in coagula, and, when there are several fibrinous remains, they cause a tuberculated appearance.

Inflammation of the placenta begins from the uterine surface, or in the substance of the organ, and presents the various stages seen in other parts, producing local or general hepatization, effusion, irregular adhesions, and secondary degenerations.

Inflammation may attack the whole, or a portion of the placenta, causing a deposit of fibrin. The effusion and compression of the tissues will vary according to the extent of the inflammation, causing more or less obliteration of the bloodvessels. In an extreme case, we find a capsule of dense lymph encasing the maternal surface, the whole is considerably reduced below the normal size, and the soft, spongy texture, is converted into a compact, splenified mass. If confined to individual lobules, the alteration will be limited in a corresponding degree. Those instances of adherent placenta which are the source of so much anxiety to the accoucheur, are probably referable to a prior inflammatory attack, gluing the afterbirth to the uterus. Professor Simpson admits the occurrence of total absorption of a placenta as one of the consequences which may result from the agglutination of the afterbirth to the uterus. A third stage of inflammation is occasionally met with in the shape of abscesses or of purulent infiltration; it is also stated to give rise to the effusion of pus between the two surfaces of the uterus and placenta. Rokitansky describes suppuration occurring here in the form of circumscribed abscesses, or of diffused infiltration and fusion. Fatty degeneration of the placenta, to which Professor Kilian and Dr. Barnes have recently drawn attention, is probably to be explained as the molecular disintegration resulting from the deposit of fibrin in the cells surrounding the villi of the chorion; and not as a primary deposit of oil within the placental capillaries, a view more fully developed by Dr. Handfield Jones,[1] in a paper on fatty degeneration. If portions of a placenta, thus degenerated, be thrown into water, "the first thing which strikes the observer," to employ the words of Dr. Barnes,[2] is, "that the tufts of villi do not expand or float out in the same way as in the healthy placenta, and, on endeavoring to separate the fragments into its component villi with needles, the extreme brittleness of the whole structure becomes apparent." Examined by a high power, "we observe: (1) that the villi are thickly studded with innumerable, minute spherules of oil; (2) the chorion is much altered; it is thickened and destitute of nuclei; (3) the walls of the vessels no longer contain nuclei, these having, in all probability, become degenerated into spherules of oil; (4) the spherules of oil are contained, some in the chorion, some in the walls of the bloodvessels, and many in the intervals or spaces between these; (5) the

[1] Medico-Chirurgical Review, April, 1853.
[2] Medico-Chirurg. Trans., vol. xxxiv.

cavities of the vessels are almost invariably free from fatty deposition: (6) the vessels are destitute of blood.

The placenta is liable to other morbid processes, of a degenerative character; the vessels may undergo cretification, and osseous deposits, of a nodulated form, are met with imbedded in the tissue; the villi of the chorion are not unfrequently found to have become converted into oval, pedunculated, serous vesicles, or hydatids, clustered together like a bunch of grapes; the cysts vary in size, from a pin's head to a filbert, and more, and they may amount to several hundred. This pathological condition has received the name of the vesicular mole; it is to the expulsion of a mass of this kind, that the fabulous accounts of women having given birth to several hundred children are attributable; the cysts having been regarded as ova, and these having been magnified into infants. Two beautiful specimens of the disease are preserved in St. George's Hospital Museum.

It is the opinion of several distinguished accoucheurs, that the vesicular mole may occur in the virgin state; in that case, the explanation we have offered is necessarily incorrect, and we must seek for it in some derangement of the uterine tissues.

Rokitansky denies the occurrence of tubercle in the placenta. The only instance on record that we have met with, is one described by M. Hardy,[1] as having been found in a phthisical female, aged thirty-five, who died suddenly, in the seventh month of pregnancy. The Cæsarean operation was performed immediately after her decease, and the fœtus was dead. Besides the ordinary appearances of pulmonary phthisis, the external surface of the uterus exhibited numerous small projections, which proved to be crude tubercles; there were none in the tissue, or on the internal surface; in the placenta, there were from eight to ten large tubercles, some as large as a pea, two nearly the size of a nut, of a whitish color, and dense; the fœtal surface of the placenta presented to the finger the sensation of a large number of miliary tubercles. The organs of the fœtus were normal.

THE FŒTUS.

The fœtus is liable to become the seat of morbid processes at every stage of its development, and in every tissue and degree; giving rise, at the earlier period of its existence, to an entire destruction of the formative nisus, or to partial arrests of development in individual parts, some of which we have had occasion to allude to in speaking of the malformations of different organs—inducing, in its later intra-uterine existence, phenomena of disease resembling those met with in the human being after birth. It remains for future inquirers to determine more accurately, not only the exact pathological character of the different lesions, but also the primary or secondary relation borne between the morbid states of the placenta and the fœtus. Many of the masses that have passed under the name of moles, have originated in a blight of the ovum; "the

[1] Archives Générales de Médecine, 1834, vol. v. p. 244.

embryo," as Dr. Ashwell describes it, "having died early, the ovum has increased in size and solidity, not by a process of growth, as in natural pregnancy, but by the effusion of coagulable lymph, from inflammation of the lining membrane. This forms successive layers over the surface of the dead ovum, giving it, eventually, a great degree of consolidation. Some of these masses exhibit no cavity, but the chorion and amnion are demonstrable, although the enveloping lymph may be one or two inches in thickness." Dr. Ashwell describes another form of moles, which does not owe its existence to conception, and may be attributable to an accumulation of fibrinous exudation within the cavity of the uterus, induced by croupy inflammation of the lining membrane. Two specimens of this may be seen in St. George's Hospital Museum (Nos. 58 and 142).

Every organ and tissue of the fœtal body may become the seat of atrophy or hypertrophy; the latter may be characterized as actual excess of one or more organs, as we often meet with in the phalanges. Numerous distortions, curvatures, even fractures, and other solutions of continuity,[1] demand the attention of the medical man immediately after the child's birth. Atrophic conditions are, generally, referable to diseased states of the placenta, which interfere with the nutrition of the child, and cause it to perish, or merely prevent its normal development. Cases are recorded in which such atrophic fœtuses have been borne the full period, though their death had taken place early in pregnancy; these, as Dr. Montgomery[2] remarks, illustrate the necessity of carefully examining into the state of the fœtal appendages as to their healthy condition or otherwise, before we venture to pronounce an opinion on the time that has elapsed since conception, merely from the size and general appearance of an ovum or fœtus shown us. In an accident of this kind, an error in judgment might seriously, though unjustly, affect the reputation of a female whose husband had died or left home shortly after conception.

The curvatures that the fœtus is most commonly subject to are, those of the lower extremities—these, as well as the dislocations of the astragalus, the elbow, and other parts that frequently come under the notice of a surgeon, are attributed to violent contractions of the uterus, or to convulsions affecting the fœtus. Of the herniæ, to which, as congenital affections, we must allude, umbilical is a frequent, and diaphragmatic the rarer form; both are the result of imperfect development of the parietes, which in each case respectively ought to completely close in the abdominal viscera. In the former, the intestines, to a greater or less extent, pass through the umbilical opening, and occupy a pouch formed by the cutaneous coverings of the abdomen; in the latter, they enter the thoracic cavity, where they displace the lungs and the heart; they commonly, though not necessarily, cause the death of the fœtus. The brain occasionally protrudes through the cranium, giving rise to hernia cerebri; this, however, must not be confounded with a tumor, which often forms on the head of the infant, simply owing to the mechanical pressure exerted upon it during parturition, and the conse-

[1] See a remarkable case that was brought before the Medico-Chirurgical Society, by Mr. T. D. Jones. Medico-Chirurg. Trans. vol. xxxii. p. 59.

[2] Art. Fœtus, in Dr. Todd's Cyclopædia of Anatomy and Physiology.

quent extravasation, and known as cephalhæmatoma. Encephalocele is described by Dr. Montgomery as, at first, a rather tense, smooth, and semitransparent tumor, giving generally a more or less distinct sense of fluctuation; in shape, the tumor is globular or oval, and frequently tapers to a neck, where it issues from the head, at which point a circular aperture can be detected in the bone, the edges of which are, in general, smoothly rounded off. The defect in the cranial bones, giving rise to this malformation, is analogous to that upon which spina bifida depends; here, there is a deficiency in the arches of one or more vertebræ, allowing a protrusion of the dura matral sheath of the canal, and the arachnoid lining, in which an accumulation of the spinal fluid takes place. The arachnoid often forms at the most projecting parts of the tumor the only investment, both the skin and the dura mater being thinned down gradually, and at last entirely lost. The fluid, in its turn, presses upon the cord, and more or less displaces it. When it occurs in the lumbar region, its ordinary site, the divided cauda equina may be seen, as Dr. Bright has shown,[1] adherent to the sac, and induces the erroneous opinion that the appearance is due to the nerves distributed over the sac being turned backwards from their natural direction. When there are several deficiences in the osseous canal, the fluid communicates between the different tumors; the entire column may be deprived of its spinous processes and their arches, so that the tumor occupies the whole region. Spina bifida is often associated with hydrocephalus.

One of the most remarkable occurrences in intra-uterine life, is the phenomenon of spontaneous amputation of a limb; this is sometimes complete—the severed extremity being entirely detached, and leaving a stump, in which the healing process is perfected;—at others only partial, the stricturing band not having cut through all the tissues. It is generally the left lower extremity that suffers; and Dr. Montgomery has demonstrated the fact of its being due to the umbilical cord being twisted round it, and not, as has been suggested by others, to gangrene, or the accidental formation of ligamentous bands. He expresses his conviction that many of the cases of apparent arrest of development may be set down to this cause, the amputated member not having been found, either from its being atrophied or buried in coagula, and from the separation having been effected at the early stages of pregnancy.

Numerous observations are recorded by authors, evidencing the occurrence of the inflammatory process in the fœtal viscera. Peritonitis, with its various sequelæ; gastro-enteritis, followed by ulceration; inflammatory lesions of the liver, pneumonia, and pleurisy; abscesses in the lungs, the thymus, thyroid glands, and supra-renal capsules, and pericarditis; have each been proved to occur in the fœtus, by Desormeaux, Billard, Simpson, Cruveilhier, Montgomery, and other pathologists, to whose works we must refer for further particulars. The same applies to the various cutaneous affections of a syphilitic, variolous, or other character. Tubercular deposits and cancerous growths have been seen, but they are both extremely rare. Nor is it compatible with our

[1] Reports of Medical Cases, vol. ii. p. 640.

limits to do more than allude to the endless varieties of monstrosities which, from the causes mentioned, or from reasons to which pathology offers no clue, affect the unborn child.

EXTRA-UTERINE PREGNANCY.

Five varieties of extra-uterine pregnancy are assumed to occur; in the Fallopian tubes, in the walls of the uterus, in the ovaries, in the peritoneal cavity, and in the vagina. We have not met with a well-authenticated instance of the last form, and the occurrence of ovarian pregnancy has also been denied by authorities like Velpeau and Kilian. The danger to mother and child is almost equal in each variety; in fact, there is only one case on record in which both have survived; this was one of peritoneal or abdominal pregnancy, which occurred to Dr. P. L. Heim, and in which the Cæsarean section was successfully performed.[1] The accident most frequently met with is the Fallopian tube pregnancy. In this case the ovum is arrested in its descent into the uterus, and the process of growth and development progresses as if it had reached its proper nidus up to the period of its discharge. The tube is distended, and its walls become hypertrophied; the changes in the maternal system, though sometimes accompanied by certain anomalous symptoms, are those met in ordinary pregnancy; there is general turgescence of the mammæ and the uterus, and in the latter organ it has long been taught that a decidua is formed, as if it contained the fœtus. Denman, Baillie, William Hunter, and Elliotson, have met with instances in which the decidua was present; other cases have been recorded by Mr. Langstaff and Dr. Lee, in which it had not formed. We have ourselves examined a preparation in St. George's Hospital Museum (No. 2,718), in which the decidua is wanting; nor does it seem difficult to understand that in one instance the sympathetic irritation should be set up by which this membrane is produced, and that it should fail in others. The development of the Fallopian tube does not keep pace with that of the ovum, and in the second or third month rupture generally takes place, the fœtus escapes into the abdominal cavity, and the mother sinks from the shock, the hemorrhage, or the peritoneal inflammation that ensues. In the majority of instances the right tube is the one affected. Rupture does not appear to be the invariable issue. In the Royal College of Surgeons (preparation No. 2,719), we find a fœtus almost completely developed, but compressed and dried, which is stated to have been removed by operation from the Fallopian tube fourteen years after gestation; the patient recovered, and lived for a long time after at Hamburg, where the operation was performed. This specimen is a good instance of what has been termed a lithopædia, a stone-child; the parts intervening between the extremities are ossified, and nutrition appears to have been thoroughly arrested. These lithopædia are sometimes retained within the uterus, as the remarkable preparation in the

[1] Rust's Magazin für die gesammte Heilkunde, vol. iii. 1817.

same museum (No. 2,720) proves, of which Dr. Cheston[1] has given a detailed account. The mother, at the age of twenty-seven, carried her fourth child to the full period, had labor-pains, but no child was born. She recovered, and died paralytic at the age of eighty. The uterus was found to contain an osseous sac adherent to the surrounding part, and resembling a middle-sized human cranium. The cyst seemed to have absorbed all the parts in contact with it, and contained a fœtus in the same position as that in utero. The brain, lungs, and liver, preserved almost their natural appearance; but there was no trace of blood, nor any remains of membranes, placenta, or umbilicus. The osseous sac, with the fœtus, weighed three pounds, one ounce, four drachms. A similar instance of thirty-two years' duration is described by Prael.[2]

The terminations of Fallopian tube gestations alluded to are not the only issue which we meet with. Adhesions form with different parts of the parietes, and the fœtus having been broken up by ulcerative disjunction, the parts may be discharged piecemeal, whereupon the cyst in which they were contained contracts, and the mother survives. In this way the fœtus has been eliminated by the rectum and the umbilicus. The above remarks also apply in the main to abdominal gestation; here the ovum, probably owing to a want of that erectile tone in the fimbriated extremity of the tube by which in ordinary pregnancy it is made to embrace the ovary when conception is effected, falls into the peritoneal cavity, the development proceeds up to a certain point, and death ensues from peritonitis, or hemorrhage, or else the fœtus is eliminated in the manner above described.

Pregnancy in the parietes of the uterus probably consists in an arrest of the ovum at the point at which the tube is inserted into the uterus; the sac, therefore, consists chiefly of the uterine tissue; but, owing to the irregular development of the organ, the process cannot run its course to the full period in the normal manner, the thinner portion gives way early in pregnancy, and death ensues, as in the other cases, from rupture and hemorrhage.

THE FALLOPIAN TUBES.

The Fallopian tubes are liable to various forms of inflammatory action. Catarrh and exudative inflammation not unfrequently cause a temporary or permanent closure of their channel, which prevents conception, and may lead to dropsical accumulations and other morbid conditions. Thus, the fimbriated extremities may become agglutinated to the ovaries, the broad ligament, or the uterus itself; or obliteration may occur at one or more points within the passage; unless the mucous membrane of the part still patent be deprived of its functions, the continued secretion will cause distension, simulating a cyst-formation; we have seen a case of dropsy of the Fallopian tube, in which the distension amounted to about five inches in diameter. At other times, according to Rokitansky, several saccular dilatations form between the separate angles and pro-

[1] Medico-Chirurgical Transactions, vol. v.
[2] De Foetu, duo de triginta Annos in Utero detento. Goettingen, 1821.

jecting duplicatures of the tubal parietes, and give rise to an imperfectly loculated pouch, which, as in the former case, may contain mucous matter of a more or less purulent character, or fluid of an heterogeneous constitution. These morbid contents are sometimes poured into the uterus, probably in consequence of the occlusion only having been effected by inspissated mucus; a less favorable issue, is rupture of the sac, and effusion of its contents into the abdominal cavity.

Cysts often affect the fimbriated extremities of the tubes; they are pediculated, and do not attain a large size. The Fallopian tubes may also be the seat of fibroid growths, carcinoma, and tubercle. Both the latter are commonly secondary to uterine disease of the same character. Dr. Lee states that carcinoma may originate in this situation; and with regard to tubercle, Rokitansky affirms that it sometimes occurs independently of uterine deposits, or in a condition of higher development. He describes it as being presented to us in the form of tubercular infiltration, and complete disorganization of the mucous membrane, which is converted into a soft purulent layer of cheesy, friable matter, that chokes up the passage, and causes the tube to be more or less swollen and tortuous, and hard to the touch.

CHAPTER XL.

THE OVARIES.

The pathology of the ovaries, important as are the questions involved in it, is still very imperfect, owing to their site, and to the absence of any secretion, the analysis of which may indicate their condition; their examination by the means at our command being often neglected from motives of delicacy, their morbid conditions are frequently not detected until an advanced stage. Many of the diseases in which the functional derangements of the system are referred to a local condition of the generative organs, are exclusively attributed to lesions of the uterus, from a definite physical examination being more practicable, and the primary derangement of the ovaries is overlooked. The stimulus which puberty gives to the development of these organs and their proper functions, is also the starting-point for their morbid conditions; before this time they are in a dormant state, and, except in connection with general diseased processes affecting the abdominal viscera, or the system at large, they rarely present any palpable lesions. It is, however, necessary to bear in mind that the ovaries may, and often are, sympathetically affected during the peritonitis of childhood, a disease of frequent occurrence. Boivin and Dugès[1] consider these inflammations a cause of obliteration of the Graafian vesicles, and a consequent source of sterility, which they may also induce by changing the position, or altering the relation, of the ovary and its Fallopian tube. Even after the period of puberty, the ovaries are exempt from many of those sources of irritation to which the uterus, from its exposure to direct injury, to contact with morbid secretion, and from the entire revolution in its functions which it suffers during and after pregnancy, would appear to be exposed; yet it has been shown by Parent Duchâtelet and others, that these local influences are not as powerful as would be assumed *à priori*; since ovarian disease has been found peculiarly prevalent among prostitutes, and frequently unaccompanied by any lesion of the external organs of generation. Of the frequent physiological congestion of the ovaries, a doubt can scarcely be entertained by any one who has watched the relation these organs bear to menstruation, independently of the direct observation of Dr. Letheby, Dr. Lee, and others, of the presence of an ovum in the Fallopian tube, and of the rupture of a Graafian vesicle in females who happened to die suddenly during the menstrual period. The almost uniform effect of a morbid arrest of the catamenia, by catarrhal or similar influences, in producing symptoms of an inflammatory

[1] Traité Pratique des Maladies de l'Utérus, &c. vol. ii. p. 509.

process in the ovaries, further corroborates this view. In the dead bodies, ovarian inflammation is rarely met with in an isolated form, but associated with affections of the uterus, or its appendages. It does, however, occur as an idiopathic disease. In the congestive stage, the organ is more or less gorged with blood, even amounting to extravasation or apoplexy, enlarged and softened. It is to the partial absorption of such extravasations, and the accompanying changes, that we must attribute the so-called false corpora lutea. These differ from the true corpus luteum in the yellow matter, from which the name is derived, being inclosed within the Graafian vesicle, while in the latter, the deposit is effected externally to the vesicle, which in the recent state retains a cavity, that only shrinks, and is obliterated after the lapse of several months.

It is in the corpora lutea of various animals that Zwicky first discovered and described certain varieties of organic crystals, since termed, by Virchow, hæmatoidin crystals, and shown, by him and other observers,[1] to be produced in the blood of all animals and of man, in extravasations occurring within the body, or when treated in a certain manner after its removal. Inflammation of the ovaries may, independently of the puerperal state, lead to suppuration. An observation of Dr. Ashwell[2] to this effect corroborates the statement of Rokitansky that, in this case, it is always limited to the follicular structures. The latter describes the coats of the follicles reddened and softened, and the cavity filled with an opaque, flocculent, sanguineous, or puriform material, which necessarily destroys the germ.

OVARIAN DROPSY.

It is to the sequelæ of acute inflammation, or to the effects of a chronic phlogistic process, that we must attribute a large number of those morbid states of the ovaries, which form so considerable an item in the list of female diseases. Both the general pathology of the organ and the etiology of the individual cases justify this remark. We particularly allude to that class of affections known under the generic term of ovarian dropsy; a disease that is characterized by the formation of cysts, which in this organ exhibit a peculiar tendency to extravagant development, a circumstance that may be attributed to the reproductive powers of the part, to the proportionally copious supply of blood, and to the entire exemption from all pressure or restraint which might limit their growth.[3] It is a disease which affects married women in a much higher ratio than single females. Mr. Safford Lee has collected and analyzed 136 cases, with a view to determining this point statistically, and finds that only thirty-seven were single, while ninety-nine were married women or widows. This fact seems to prove that the ovary undergoes a different excitement

[1] See an article in the British and Foreign Review, October, 1853, on Albuminous Crystallization, by E. H. Sieveking.
[2] On the Diseases of Women, p. 625.
[3] See Dr. Hodgkin's Lectures on the Morbid Anatomy of Serous and Mucous Membranes, vol. i. p. 242.

in the discharge of an ovum resulting from impregnation, than when this takes place simply as a concomitant of the catamenia. Another point established by the same author has reference to the age at which women are most liable to the affection. He shows that it is erroneous to assume dropsy of the ovary to prevail during the decline of life. One hundred and forty cases are distributed in the following manner:—

From 15 to 20.	20 to 30.	30 to 40.	40 to 50.	50 to 60.	60 to 70.	70 to 80.	Age not specified.
3	37	45	26	19	3	2	5

This, as well as the preceding numbers, satisfactorily proves that there is an intimate connection between the morbid impetus inducing the disease and the reproductive process. In the second table, we also find a feature which distinguishes the former from the cancerous diathesis, for while the climax of ovarian dropsy is between the thirtieth and fortieth, that of carcinoma is demonstrated to be between the fortieth and fiftieth years. Dr. Hodgkin is of opinion that there is an hereditary predisposition in some females to the production of ovarian cysts, but that we rarely find a simultaneous affection of the same kind in other parts of the body. It is not often that we see both ovaries affected; the right, according to the statistics of Mr. Lee, being the more frequent seat of the malady; of ninety-three cases, collected by him, fifty occurred on the right, thirty-five on the left side, and eight on both sides.

Ovarian dropsy presents various forms characterized by features well known to the practitioner, and equally distinct in their pathological relations. The cyst-growths which constitute the disease are simple or unilocular, compound or multilocular, and complicated or cancerous.

Simple cysts are globular sacs, containing fluid, formed of an envelop with a single undivided cavity, and inclosed in the ovary or external to it. They are solitary or numerous, and we occasionally find an ovary scarcely exceeding the normal size, in which the stroma has disappeared, and is replaced by small cysts of this description, varying in size from a pin's head to a pea. In this case they are, probably, always the result of a distension of the Graafian vesicles by a morbid increase of their contents, which, at the same time, necessarily undergo a change in their constitution. The number of the Graafian follicles is stated to amount to about fifteen; where the number of cysts considerably exceed this proportion, we may perhaps be justified in attributing them to an independent germinative power, though it suggests itself, that if the view be correct that an ovum is discharged at each menstrual period, either anatomists err in their statement, or another view is requisite to account for their successive formation; upon this theory, there would be no unfair assumption of an identity between the processes. Another argument in favor of the doctrine that the simple cysts result from an hypertrophy of the Graafian vesicles may be derived from the well-known fact that they are often found to contain evidences of morbid germination, in the shape of hair, teeth, and bones, not referable to impregnation. They may attain a very considerable size, so as to hold

several gallons of fluid; the envelop then presents a leathery appearance, varying in thickness from a quarter of a line to several inches, and their fluctuation becomes a very manifest diagnostic sign. A specimen of this kind is the preparation marked X 16, in the Museum of St. George's Hospital, which was connected with the right ovary, and in the parietes of which several well-developed teeth are inserted,[1] as into the alveoli of the jaws, their crowns projecting freely into the cavity. They more commonly, however, do not reach this magnitude, are of very

Fig. 330.

Incipient cyst-formation. The ovary is represented of the normal size.

slow growth, and resist medicinal agents. The fluid they contain varies as much in color and constitution as in quantity; it is a clear, limpid, straw-colored, highly albuminous fluid; or it presents a viscid, glairy, more or less opaque character; or we find it of a coffee color, or greenish, with a large quantity of oily matter floating on the surface. In the latter cases, the microscope demonstrates the presence of blood-corpuscles and cholesterin plates. During life, paracentesis often is followed by discharges of several kinds of fluid at different periods of the operation; this can only occur in consequence of our having to deal with a case in which there are several cysts, which are penetrated by the trocar or give way spontaneously.

Simple cysts, that form on the surface, like those produced in the vicinity, may be altogether a new formation, though it is to be remembered that in the advanced stage of development the relation of the cyst to the ovary is considerably altered from what it presents at earlier periods. Pedunculated cysts are always small in size, possess thin coats, and their contents are transparent. They evidently possess a character distinct from those previously spoken of.

Multilocular cysts are growths possessed of a power of self-multiplication, which is as surprising in its features as it is characteristic of the heterologous source of their development. They also form more or less circular tumors, occasionally nodulated on the surface from the projection of some of the contained secondary cysts. The opportunity is

[1] We do not deny the occasional anomalous formation of bone, teeth, and hair, in cysts contained in other situations besides the ovary, but the prevalence of this phenomenon in the latter indicates a peculiar tendency.

rarely afforded of examining the disease in its early stages, as it does not prove fatal until it has attained an enormous size; still, the various appearances it presents are reducible to three classes, which Dr. Hodgkin was the first satisfactorily to demonstrate. In the first variety, we find that incision into the enlarged ovary, instead of exposing a single cavity, displays numerous chambers, each filled with smaller cysts, which, in their interior, commonly exhibit a tertiary formation of the same kind. The septa are probably the result of rupture from over-distension of cysts in the earlier stages. The cysts are permanently attached to their respective matrix, bloodvessels ramify freely over them, and there is a continuous and uniform epithelial investment. Their contents are commonly of a viscid, glairy, albuminous character, varying much in color, yellow, green, blue, red, and grumous, or transparent, probably owing to accidental local irritation and the admixture of the inflammatory products. The microscopic appearances of the

Fig. 331.

A multilocular ovarian cyst, removed from a female, æt. 29, during life, by Mr. J. B. Brown. Septa form larger compartments, in which there is a secondary and tertiary growth of cysts. The tumour weighed 11 lbs. 3 oz.

contents will vary accordingly; but we have found what may be considered the genuine cyst-products to consist of granular cells of circular form with well-defined outlines, conveying the impression that the fluid itself was a germinating nidus. The celloid particles vary in size from $\frac{3}{10000}$ to $\frac{7}{10000}$ of an inch; they float in a fluid blastema, are colorless, and contain one or more granular nuclei; there are also corpuscles that are identical with blood-corpuscles, though not contained in vascular channels. The walls of the cyst consist of delicate fibroid tissue, covered by a layer of cells, and delicate cells may be seen imbedded in the tissue.

The second variety of compound cysts is characterized by the development of clusters of pedunculated cysts from the inner surface of the primary sac. "It sometimes happens that the number of cysts forming the cluster is so great in proportion to the space they occupy, that, like trees too thickly planted, they interfere with each other's growth; their

development is more or less limited to an increase of dimension in length, yet, as their free extremities are allowed to diverge, we sometimes find the slender peduncle gradually dilating into a pyriform cyst, at other times the dilatation does not take place till near the extremity of the peduncle, and it thus produces a cyst more nearly resembling a grape or currant."[1] The pedunculated cysts may grow directly from the matrix and form a common footstalk; sometimes they are very vascular, at others but feebly organized, and may become entirely deprived of vitality, in which case they prove a frequent source of irritation to the containing cyst. This induces inflammatory secretions of an inorganizable kind, of a thick, yellow, grumous appearance.

In the third variety, Dr. Hodgkin describes the secondary cysts as distinguished by the breadth of their basis and by a flattened form; they are collected in clusters upon the external cyst, and produce a circumscribed and more or less considerable thickening of the parietes. The tertiary cysts found within them present the same broad sessile character, and both contain, "sometimes a mucous, at others a serous," secretion. We should be inclined to class the first and third forms together as mere accidental variations of the same type. This remark almost applies to the three varieties, since the fact of their being frequently combined in the same ovary indicates the same fundamental morbid tendency and impulse.

Rokitansky describes a form of compound ovarian cysts, which he regards as identical with alveolar cancer, though peculiar to the ovary. He states it to be an accumulation of fibrous sacs, containing a glutinous matter, which diminish from the circumference towards the interior, and especially towards the base of the morbid growth, so that this presents a condensed alveolar mass, of a distinctly malignant character.

The complicated form of ovarian dropsy is that in which the cyst formation is, as it were, engrafted upon, or associated with, other diseased states of the organ, such as hypertrophy, fibrous tumors, or carcinomatous growths; this is a feature which becomes of practical importance in reference to the influence of curative means, and the question of extirpation. The latter is also closely affected by those secondary inflammatory processes which the enlarging ovary is apt to induce; peritonitis very commonly results, by which adhesions of the ovary to the surrounding tissues are effected; and while the pressure of the enlarging tumor materially interferes with the functions of the surrounding viscera, the bladder, the intestines, the kidneys, the diaphragm, and respiratory organs, it also exerts a dynamical effect in producing ulceration and perforation of the colon or bladder. In the earlier stages, ovarian tumors prove serious impediments to parturition.

FIBROUS GROWTHS.

These affect the ovaries as primary formations evolved in the substance of the organ and closely resembling those which we have described as occurring in the uterus; or, as a secondary development,

[1] Hodgkin, l. c. p. 233.

accompanying the evolution of ovarian cysts. The fibrous tumor is developed in the tissue of the ovary, presents a globular form, and though of slow growth may attain an enormous size. The largest one on record occurred in the practice of Dr. Simpson, and weighed fifty-six pounds. The outline of the tumor is well defined; it is commonly supposed to be very scantily provided with bloodvessels, against which the appearance of some injected specimens, contained in the Museum of St. George's Hospital, appears to militate; one (No. 145), that occupies the greater part of the organ, actually exhibits greater vascularity than the surrounding normal texture. Like the tumors of the same class in the uterus, they are here also found to give rise to small cysts in their interior. Fibrous tumors of the ovary occasionally exhibit a tendency to so-called ossification, which consists in the conversion of a portion of its tissue into calcareous matter.

Tubercle occurs so rarely in the ovaries that the majority of morbid anatomists are either silent on the subject, or even, as, for instance, Rokitansky, deny its development in this locality altogether. Louis, however, established the fact of these organs not being exempt, as he has observed a small quantity of tuberculous matter in two instances.

Malignant disease assumes several forms in the ovary, and though its frequency has been exaggerated by Boivin and Dugès,[1] who state that it is more common than carcinoma of the female breast, and only less so than that of the uterus, all modern pathologists are agreed as to its being by no means a rare affection. It appears as scirrhus, encephaloid, hæmatoid, melanotic, and alveolar cancer, either as an isolated growth, or in the infiltrated form. "Cancerous matter," to use Dr. Walshe's words, "especially the encephaloid, is more commonly discovered in the ovary as an addition to some other morbid formation than as the sole disease of the organ; multilocular cystoid productions are those to which it is most frequently superadded; in much more rare instances a multilocular cyst pre-exists. Under such circumstances, the cancerous deposition goes on in connection with the walls of the cysts, and exhibits all the varieties of pedunculated and sessile forms. When thus formed in the walls of cysts, encephaloid frequently coexists with fibrous, cartilaginiform, calcareous, or ossiform infiltration of those walls."

Ovarian cancer runs a rapid course; it is generally limited to the ovary of one side, and, as it extends, is liable to induce inflammation of the surrounding parts, causing adhesions, which will fix the tumor in the pelvis, or attach it to the viscera, occupying a higher level according to the period at which they form. Cancer occurs more frequently at an earlier period of life in this organ than in the uterus; Dr. Walshe has found forty-one years the average age at death; but it has been met with even before puberty.

THE MAMMÆ.

Diseases of the mammæ are essentially diseases of the adult female—we must not, however, overlook those cases which occur in the male sex and in infant life.

[1] Traité Pratique, &c. vol. ii. p. 654.

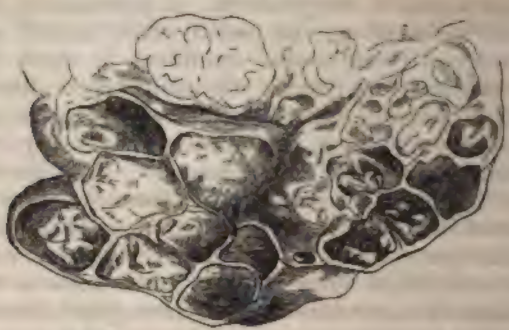
At birth, the breasts of infants of either sex are very commonly swollen and somewhat indurated; they contain a whitish serum, which induces nurses to employ friction for the purpose, as it is termed, of rubbing the milk away. The result of this proceeding is frequently to induce inflammation and abscess in the part; which, however, may also occur spontaneously, and even become chronic. At the period of puberty, the gradual development of the glands in the female serves to mark their ultimate function, and to define the character of the sex. It is now that irregular evolution is exhibited as an atrophic, or an hypertrophic condition of the whole gland, or of the nipple only. The proper tissue of the gland; as well as the surrounding fat, and cellular and cutaneous textures, may be separately or coincidentally involved in each case. The hypertrophy sometimes attains a very considerable size in unmarried females, and appears to be an indication of a generally precocious tendency. Commonly, both breasts are affected; but occasionally, one is inordinately developed. A temporary enlargement of the gland very commonly accompanies menstruation; it may occur periodically, even long after the cessation of this function, as in an old lady of eighty-five, who was under our care, and who was subject to this phenomenon regularly every month. A permanently hypertrophic state is induced by lactation; the period which directly or indirectly gives rise to many of those morbid conditions to which the gland is liable. Inflammation is one that in the acute form is almost limited to this period. It produces congestion in one or more parts, accompanied by swelling, interstitial effusion, condensation, and finally, if the disease be not arrested, suppuration and abscesses. The glandular texture itself, or more frequently, the intercellular tissue, is the primary seat of lesion; the lacteal secretion frequently continuing during the inflammatory process, and even after a chronic state of induration and enlargement has been established, which has led to the removal of the organ. When the inflammation is confined to the true gland structure, the resulting tumefaction is irregular and lobulated, and deeper seated than when the interstitial or cutaneous tissues are mainly involved. When suppuration is established, it may be limited to one spot by adhesive inflammation, and the abscess be evacuated by pointing, as it usually does, near the nipple; or burrowing sinuses form, which may extend to a considerable distance. During lactation, large accumulations of milk frequently distend the entire system of ducts, or a single portion; in the latter case, a fluctuating tumor may result, which will scarcely disappear without surgical interference. It is stated, that as much as ten pints of milk have been evacuated from a swelling of this description. It appears to be generally owing to an atonic state of the mammary ducts, similar to the condition of the efferent channels in the nipple, causing a non-retention of the secretion. The lactiferous tubes are occasionally found to contain sebaceous-looking matter, phosphatic concretions, and other products, which have been attributed to the effects of chronic inflammation (College of Surgeons, Nos. 2,743, 2,744, 2,747, 2,748); but unless there is a coincident change in the coats of the ducts, it is probable that these matters are the residue of an effusion of

milk, which has been long retained, and in which a partial absorption has taken place.

Adventitious growths are very frequently met with in the mammary glands, presenting the characters of benignant and malignant formations, and deserving careful attention on account of the pathological as well as the practical interest that attaches to them. Much difficulty is often presented in determining the diagnosis during life, and it is only since the microscope has been brought to bear upon an analysis of these mammary affections that a clear light has been shed upon them, which has already borne fruits in practice.

Encysted tumors present two great classes, those dependent upon an expansion of the ducts, and those resulting from a new growth in the fibro-cellular or adipose tissue; a state of things closely analogous to what we meet with in the kidney. To the former we have already alluded. The latter occurs as an isolated globular or oval and more or less movable cyst, or there are numerous growths of this kind, varying in size from a pin's head to a hen's egg. The inner surface is smooth,

Fig. 332.

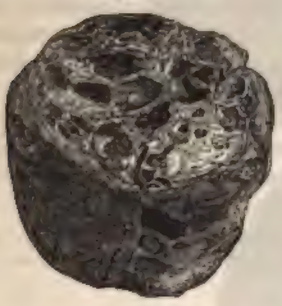


or it presents a broad-based lobulated cauliflower-like growth or warty excrescences, and the substance of the surrounding gland is indurated and atrophied. These cysts may also occasion a retraction of the nipple, a point of practical importance, as this is often looked upon as pathognomonic of cancer. The transverse section exhibits a double sheath, one proper to the cyst, the other the result of condensation of the adjoining textures. The contents of the cyst may be fluid or solid; the former presenting either a limpid and opalescent, non-albuminous character, or a grumous, brownish, more or less sanguineous appearance, in which case it is highly albuminous; the latter approaching the character of fibroid deposits, being composed of a pale, firm, compact substance, traversed by undulating fibrous lines, which imperfectly divide it into lobes of various sizes or shapes. These cysts have been termed cysto-sarcoma, or serocystic growths, by Sir Astley Cooper, and Sir Benjamin Brodie. Mr. Birkett[1] has found that they contain imperfect gland-tissue, and that the

[1] The Diseases of the Breast, and their Treatment. London, 1850, p. 64, seqq.

intra-cystic growth is invested by a reflection of epithelium from the cyst-wall. His views on the subject may be thus expressed: Certain collections of a plastic fluid take place in the areolar tissue of the gland, a closed cavity is formed, lined with tessellated epithelium, and the intra-cystic growths, being within the sphere of nutrition of the mammary gland, present more or less resemblance to the latter; hence, they may be regarded as imperfectly-developed gland-tissue, which offers no analogy to carcinoma, either in its local appearance or in its effect upon the sys-

Fig. 333.



Example of cysto-sarcoma, from the breast. At *a*, the cysts many; distinctly lined by a secreting membrane, and filled with a glairy fluid. At *b*, a section made on another plane; cysts less numerous.

Fig. 334.

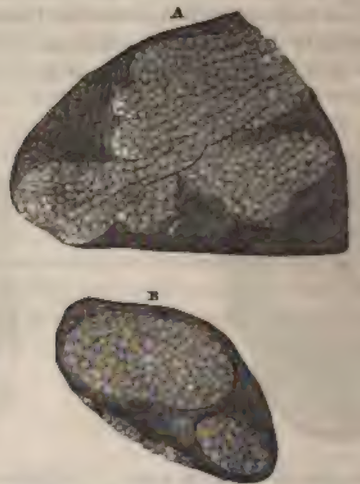
Cysto-sarcoma simplex, from the neighborhood of the mamma. One large cyst, *a*. *b*. The solid part of the tumor; a simple stroma.

tem. While we would not altogether deny the conclusions at which Mr. Birkett has arrived on this point, we should be disposed to question the secondary formation of gland-tissue in a previously-existing cyst, and, where we meet with an encysted growth of this kind, include it under what he has appropriately termed lobular imperfect hypertrophy (l. c. p. 124); for although the tumors of this description ordinarily remain in connection with the proper gland-tissue, they sometimes appear to be altogether isolated, and a capsule of condensed cell-tissue easily simulates a genuine cyst.

In a small tumor of this description, removed from the breast by operation, on the supposition of its scirrhoid nature and of its being unconnected with the gland, which we had an opportunity of examining, the microscope revealed well-marked ducts and lobules, in no essential feature differing from ordinary mammary tissue. We have since repeatedly examined the structure of mammary tumors, which were regarded as malignant, and found them to consist of follicular structure filled with epithelial growth. Mr. Birkett describes this form of tumor as presenting to the naked eye a granular appearance of a white, rosy, or red color, dependent in a measure upon the time it has been exposed to the air; it is lobulated, divisible into the most minute lobules, attached by a prolongation to the breast, and invested by a fibro-cellular envelop continuous with the proper fascia of the gland. The lobules are connected by common areolar tissue, and the growths vary in size from that of a marble to that of a child's head.

The genuine hydatid cyst, containing the echinococcus, occurs in the female breast, and may be recognized by its well-known features. The tumor itself grows imperceptibly, causes no annoyance to the patient,

Fig. 335.



Lobular hypertrophy of mamma. A. Section, showing the entrance of duct. B. Cross section, resembling cystic disease.

is firm to the touch, and contains clear fluid, in which the microscope detects the tentacula of the echinococcus, the animalcule itself being attached to the internal wall of the cavity.

Many so-called fibrous tumors of the breast are to be considered as

Fig. 336.

Fig. 337.

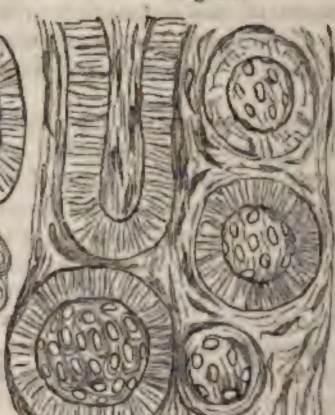


Fig. 338.

This series of diagrams represents microscopic sections of a simple tumor removed by operation from the female breast; consisting mainly of hypertrophy of the fibrous structure of the gland, with enlargement of the included ducts and their epithelial linings. *c.* Section of the epithelium from one of the tubes. *b.* Group of epithelial cells from the same. *a.* The same after the addition of acetic acid.—Bennett.

Mr. Birkett has demonstrated, as belonging to the same class as that just considered; and though he does not deny the existence of an hypertrophied condition of the fibrous tissue, he has found the elementary gland tissue to preponderate in all the cases called fibrous which he has examined. This accords with the observations of other pathologists, who either deny the occurrence of fibrous, cartilaginous, and osseous tissues in the mamma, or adduce only doubtful or solitary instances. Sir Astley Cooper removed a tumor from a young woman, aged thirty-two, the larger portion of which had the appearance of the cartilage supplying the place of bone in the young subject; the remainder being ossified. Professor Müller states that he met with a case of enchondroma in the organ.

Tubercle has not been met with in the mamma. Cancer affects the mamma more frequently than any organ of the body; it obeys the same laws as to the period of life in which it most flourishes as we have traced when speaking of uterine cancer. The analysis of one hundred and forty-seven cases[1] exhibits a marked preponderance in the fifth decennium of life, as the following table, given by Mr. Birkett, shows:—

From 1 to 10 years	1 case.	From 50 to 60 years	29 cases.
" 10 " 20 "	8 cases.	" 60 " 70 "	10 "
" 20 " 30 "	11 "	" 70 " 80 "	2 "
" 30 " 40 "	32 "	" 80 " 90 "	7 "
" 40 " 50 "	51 "	" 90 " 100 "	1 "

Of one hundred and sixteen cases, seventy-nine were married women, and thirty-seven single; of fifty-five married women, forty-seven were prolific, many of them having borne several children, and only eight were sterile. Sir A. Cooper knew a woman with this disease who was pregnant seventeen times. These numbers must not, however, be taken as absolute indications, but only approximatively; it is to be remembered that the proclivity to cancer being greatest between forty and fifty, the correct ratio of married and unmarried females could only be calculated by knowing the total numbers of these two classes. Dr. Walshe shows that the left side is more frequently affected than the right; of one hundred and two cases, fifty-nine were limited to the left, thirty-nine to the right breast, and in four only were both implicated.

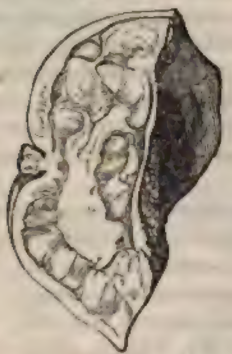
All the varieties of carcinoma have been met with in the breast; scirrhus is, however, by far the most frequent form in which it occurs primarily; the encephaloid variety may be primary, but is more commonly engrafted upon the former; the colloid form is the most rare. When associated with other cancers, that of the mamma is stated by Dr. Walshe to be invariably primary, except in those rare instances when the disease spreads from the lymphatic glands or superjacent skin.

Scirrhus occurs in the form of a hard, lobulated tumor, imbedded in the adipose tissue of the gland, causing adhesion to the skin, and retraction of the nipple; at first, somewhat movable, but soon becoming firmly adherent to the subjacent parts, and involving more or less of the gland-tissue, the thoracic muscles, and the adjoining glands. Instead, however, of occurring as an isolated tumor in the first instance, it may, from the

[1] Birkett, l. c. p. 218.

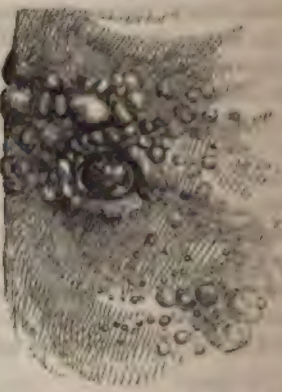
commencement, appear as an infiltration of the various structures of the part; it will then be ill-defined, sending out branches into the adjacent tissues, and in that case involving in its mass the lacteal tubes and

Fig. 339.



Carcinoma of the breast, bisected. The figure of the tumor, with its effect on the gland and nipple shown.

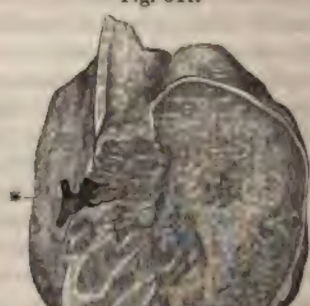
Fig. 340.



Carcinoma, secondary. An example of the numerous nodulated tumors, which often form in the cicatrix of the former growth. One is ulcerated in the site of the mammilla.

lymphatics. These become contracted and flattened into many bands, which give a peculiar appearance to this form of mammary cancer. Dr. Walshe has repeatedly seen similar structures when cut across, exhibiting an obvious bore, and he has succeeded in detecting the character in some

Fig. 341.



Section of a large, hard, cancerous tumor, from a woman æt. 60, imbedded in the breast, exhibiting a pale dull-grayish basis, shaded with light pink, and intersected in every direction by short wavy lines, like bundles of white fibres, which mingle together in a close irregular network. This fibrous structure is most distinct about the centre of the mass, its exterior appears more homogeneous. *. The retracted nipple.—St. Barthol. Museum, xxxiv. 14.

seen lengthwise; but he has never noticed these forms except in mammary cancer. Scirrhus is not at first accompanied by pain; hence its existence is often accidentally discovered when it has already reached the size of a marble or a pigeon's egg. Ulceration of the skin, in the

vicinity of the nipple, supervenes; the edges of the sore are raised, everted, and puckered; a purulent, ichorous fluid is secreted, from a bluish-red, eroded surface, offering a faint and fetid odor; bleeding often ensues, and the patient sinks from exhaustion. The amount of pain suffered appears to depend upon the nervous constitution of the individual, since it does not present any uniform proportion to the extent of the primary affection, or of the secondary ulceration; one of the largest scirrhous tumors that has fallen under our observation, followed by extensive ulceration and frequent hemorrhage, gave rise to no bodily pain from first to last; while it is a matter of daily observation that intense lacerating pains in the breast and arm of the affected side accompany tumors whose extent does not warrant the supposition that any direct irritation of the nerves can take place. Scirrhus generally attains the size of an orange or a man's fist, and more; it passes through its various stages with more or less rapidity, averaging about three years altogether; the ulcerative stage once commenced, the powers of the system are soon broken. Sir A. Cooper states that the period of growth lasts from two to three years, and that, after it has attained its full development, the time in which it proves fatal varies from six months to two years. The older the individual at the time at which it first appears, the slower its subsequent growth; hence, the practical rule followed by Sir A. Cooper, that, in advanced life, surgical interference is neither humane nor scientific. The axillary lymphatic glands are commonly swollen, hard, and infiltrated with the same carcinomatous product as the mamma; their affection appears to be coincident with the implication of the skin at the primary seat of injury. The pectoral muscles, the ribs and costal cartilages, are also found secondarily involved to a greater or less extent; a secondary affection of the pleura and the lung is not unfrequent. Towards the termination of the disease, from direct interference with the venous circulation, œdema of the extremity of the affected side is liable to supervene.

The encephaloid form of the disease occurs earlier in life, and generally runs a more rapid course than the one just considered. It is much less frequent, and, though sometimes primary, is commonly met with as a complication of scirrhus. Moreover, it presents a less defined margin; according to Sir Astley Cooper, it is difficult to say where the diseased structure terminates, and where the healthy structure commences; the base of the tumor is, therefore, diffused among the healthy cellular membrane, or other parts, where it shall happen to be situated; another diagnostic sign, as pointed out by Sir A. Cooper, is that the disease may advance even to suppuration and ulceration without the glands of the axilla becoming at all affected.

THE MALE MAMMA.

The male breast is occasionally the seat of non-malignant and malignant growths. We have ourselves met with an instance, in a gentleman aged twenty-one, of the former, which, to the touch, closely resembled one of hypertrophy of the mammary gland; it was of the size of a shil-

ling; felt semi-cartilaginous, as if composed of lacteal ducts, and was adherent to the skin of the nipple. The individual had perceived it six weeks before applying for advice; it gave no pain, and four months later we were informed that it had almost disappeared without any active treatment being pursued. This is in accordance with the structure of the male mamma, which Sir A. Cooper has shown to resemble the female gland, though in a rudimentary state. Nor could we otherwise account for the well-authenticated cases of the secretion of milk by men. Mr. Birkett gives delineations, showing the male gland to have all the essential elements requisite for the performance of the function. Mr. Stanley[1] relates the case of a man, aged forty-five, who was affected with cancer of the right humerus, secondary to cancer of the right breast. Cruveilhier states that three cases have come under his observation, one of which is delineated in his atlas.[2] In the College of Surgeons (prep. 2,791), there is the section of the breast of a man with a very vascular ulcer, five inches in diameter, probably originating in a lens-shaped, hard, cancerous tumor, or a degeneration of the skin and mammary gland. The monographs on diseases of the mamma also contain records of simple cysts, compound cysts, and encysted tumors, occurring in the male breast; but they belong to the mere curiosities of medical experience.

[1] A Treatise on the Diseases of the Bones, by Edward Stanley, F. R. S., 1849, p. 255.
[2] Anatomie Pathol. Livr. xxiv.

THE PATHOLOGICAL ANATOMY OF THE JOINTS.

CHAPTER XLI.

DISEASES OF THE JOINTS.

Malformations.—In cases of defective development, some joints may be quite absent, the bones may be united by congenital anchylosis; or, in a less degree of imperfection, they may be incompletely formed, the ligaments sometimes being partly or altogether wanting even when the rudimentary extremity of the bone is covered with cartilage. On the other hand, supernumerary joints exist, both when the number of bones is natural and when it is excessive.

Inflammation of the Synovial Membrane.—This may arise as a primary disease spontaneously, from cold, from injuries, from localization of the rheumatic poison, or from that of syphilis, or gonorrhœa. It also occurs as a secondary affection, excited by disease of the cartilage, or of the subjacent osseous tissue. It may be acute in various degrees, or chronic. It is rare in young children, less so about the age of puberty, and very frequent in adults. Before we describe the morbid changes, we must advert to two points in the anatomical arrangement of this membrane, which are of much importance. The synovial are commonly, and no doubt justly, classed with the serous membranes, and are described to form shut sacs, just as these are. Dissection, however, fails to trace the membrane over the free surface of the cartilages, and microscopic examination confirms its absence, except in the fœtus. In these unused joints the cartilaginous surface is found quite smooth and even, and covered by a layer of delicate epithelial scales, such as line the surface of the synovial membrane when it passes over the ligaments. In the articulations, however, of adults, not only is the epithelial layer absent, but the surface of the cartilage is slightly irregular, as if somewhat worn. Our own examinations have convinced us of the general correctness of these statements, given by Dr. Todd and Mr. Bowman, but we must also mention that another high authority, Mr. Toynbee, is of a different opinion, and believes that he can demonstrate the existence of the synovial membrane in the adult, by detaching an exceedingly delicate layer from the cartilage, which, he states, does not contain any of the cartilage-cells. This, we think, is a film of the cartilage itself,

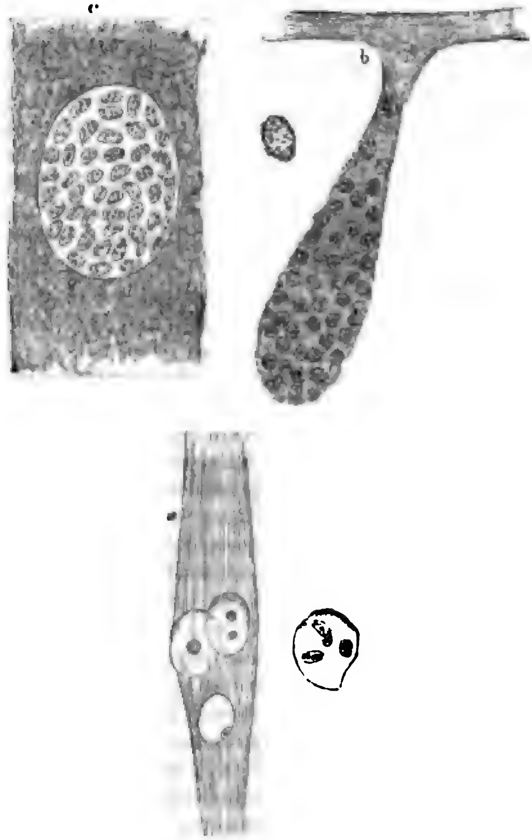
only so thin that it cannot include the cells. The other point we wish to notice is the existence of a set of remarkable vascular processes, covered by a delicate epithelium, upon the free projecting margin of those synovial folds which advance into the cavities of joints. Mr. Rainey, by his discovery of these, has confirmed the anticipation of Clopton Havers, that those synovial folds fulfil, in some measure, the function of glands, being particularly concerned in the formation of the synovia. It is, we are convinced, from these vascular processes that bloodvessels first enter the false membrane formed by exuded lymph.

The results of *acute synovitis* are the following: More or less injection of the vessels, which in one instance, related by Sir B. Brodie, were so distended with blood that, "throughout the whole of its internal surface, except where it covered the cartilages, the synovial membrane was of a dark red color," like the conjunctiva in acute ophthalmia. Effusion of serous fluid, which may be so great as to lead one to suppose that the sac is filled with solid matter. Effusion of lymph, forming flakes all over the synovial surface, and not upon the cartilages. In severe cases suppuration may occur. If the disease advance unchecked, ulceration of the cartilages is very prone to occur; the exuded lymph then forms villous or fringed processes, which are in contact with the ulcerating part of the cartilage, and probably both promote the destructive process, and aid in removing, by absorption, the disintegrating tissue. We shall return to this point again under the head of ulceration of cartilage. Under judicious treatment, the whole of the fluid will be reabsorbed, and the joint return to a perfectly healthy state. If, however, much solid exudation is present, its absorption will be more difficult, and some amount of swelling and stiffness of the part will still remain.

"Chronic synovitis," Sir B. Brodie says, "causes an increased secretion of fluid, but does not in general terminate in the effusion of coagulable lymph, or in thickening of the inflamed membrane." Fibrinous matter is, however, if the disease continue long, or often recur, effused either on the inside or outside of the synovial membrane, and becoming gradually organized into a fibroid tissue, thickens its substance and renders it sometimes firm and gristly. A preparation in the Museum of St. George's Hospital, shows the synovial membrane of the knee-joint so altered in this way as to be nearly an inch thick. It may be difficult, if not impossible, to detect the presence of fluid in the cavity of a joint which is in this state. Serous effusion, to a considerable amount, sometimes takes place in the synovial sac, without any manifest inflammation. The affection is analogous to hydrocele, and belongs to the class of passive dropsies. Its causes are generally obscure. When abscess occurs in a joint, the pus is commonly mixed with more or less of synovial fluid, and flakes of lymph, and is sometimes quite of a sea-green color. There is, also, sometimes, suppuration outside the joint, the color of the muscles is altered, the periosteum and the osseous structure in the vicinity are injected and inflamed. A rapid effusion of pus into the synovial cavities of joints, not unfrequently occurs in phlebitis, puerperal fever, erysipelas, and in cases of contamination of the blood by some morbid matter. In one instance of this kind occurring after a thecal abscess in a finger, we found the synovial membrane forming the

nargin of one of the ligamenta alaria of the knee-joint manifestly in-ected, and fringed with a number of various-sized villous projections. These consisted of a fibro-homogeneous, granulous substance, imbedding numerous glomeruli. A layer of similar matter was spread over the whole of the synovial surface, which was not injected with blood. The cartilage was ulcerated in some part of its extent, its surface rendered

Fig. 842.



From a case of secondary depot in knee-joint, the same as described in the text, in the next page.
(c) A cartilage-cell, immensely hypertrophied, lying in fibrous stuff.
(b) A villous process springing from the synovial membrane.
(a) A strip of fibrous tissue containing three enlarged cartilage-cells—one is also figured separately.

irregular by superficial erosions, and its texture altered to a lax fibroid stuff. The cartilage-cells in these parts were most remarkably changed, containing sometimes 20–25 celloid masses in their interior, instead of the two or three which they might contain in their normal state. The inter-cell substance was entirely deprived of its natural consistence; it broke down under slight pressure. The following description of the condition of a joint which had been long in a state of suppuration is so

faithful and life-like that we cannot forbear transcribing it. Rokitansky says: "The quantity of purulent fluid effused into the cavity of the joint is generally considerable, and the capsule is, consequently, much enlarged; the synovial membrane is lined with a firm, shreddy layer of lymph, which is dissolving into pus, and a soft, purulent precipitate, which can be easily removed, adheres to the cartilages." The term precipitate, which is here employed, is worth noticing, as Rokitansky states most strongly, in a previous paragraph, "that no exudation is deposited on that portion of the synovial membrane which covers the cartilage;" if any is found there, it is to be regarded as a "*secondary precipitate*" from the general exudation. This circumstance strongly confirms our view of the disposition of the synovial membrane. The layer of fibrin, lining the synovial membrane, "is opaque and lustreless, its surface is rough, and serum is infiltrated, and blood in small spots extravasated through its tissue, as well as through that of the fibrous capsule of the joint, and neighboring cellular structures. As the disease advances, the infiltration and thickening of the neighboring structures increase, they become filled with a gelatinous, lardaceous, white product, in the midst of which fibrous tissues, capsules, ligaments, or aponeuroses, can no longer be recognized. Here and there, in the mass, there are cavities of different dimensions, the lining of which is vascular, spongy, and granulating, and the contents purulent. The muscles near the joint are pale and flabby, infiltered and attenuated. At length the infiltration reaches the subcutaneous cellular and adipose tissues, and the integuments become fixed to the disorganized structures beneath. The diseased joint then presents the following external appearance: it is swollen, and always more or less bent; it feels everywhere soft and flabby, or in some spots flabby, in others firm, elastic, doughy, and at the same time tuberculated; the integuments over it are tense and pallid, leuco-phlegmatic, or they are traversed by varicose veins.

At length ulceration commences, and advances in various directions. "Externally, the capsule ulcerates in one or more spots, and then the soft parts adjoining it. In some instances large openings form in the capsule, and connect the joint with ulcerated cavities in the soft parts; in others, mere sinuses are formed; but in either case they open externally through the skin, and occasion and maintain a discharge of the contents of the joint. Internally, the inter-articular cartilages and the ligaments ulcerate, the cartilage covering the bones, when brought into contact with the matter, is destroyed in the way that has been mentioned, and the ulcerative inflammation attacks even the bones, if they have not been involved already. The cavity of the joint appears like a cloaca, surrounded with a gelatino-lardaceous mass; the integuments covering it are of a dark red hue, and are especially discolored at the orifices of the sinuses. The joint contains pus or sanies of an offensive odor and variously discolored; the repeated hemorrhages which take place when there is acute caries of the bones, very frequently giving it a red or brown tinge; the ligaments ulcerate, and the cartilages separate partly, or entirely, from the bones; the osseous surfaces are laid bare, their compact wall is destroyed, and the spongy tissue is exposed,

infiltrated with pus, and ulcerating, and surrounded on all sides by osteophytes of various shapes; remains of the fibrous structures of the joint, pieces of loosened cartilage, and of necrosed bone, are mixed with the matter discharged from the joint. The soft parts, and the entire bones belonging to the diseased joint, are wasted, most of the fat is absorbed, the muscles are remarkably blanched and thin, and the bones, being generally in a state of eccentric atrophy, are soft and fragile. More or less quickly after the disease has reached this stage, spontaneous dislocations, as they are called, ensue." The exudative product of inflammation, in some instances, is, according to Rokitansky, converted into tubercle. This occurs when there is a great amount of general tubercular disease. The articular extremities of bones are sometimes affected by tuberculosis simultaneously with the synovial membrane, sometimes before it.

Inflammation may attack the cellular tissue *around* joints, causing effusion of coagulating fluid and consequent swelling, with subsequent formation of pus. One or more spots only may be affected, so that small, local deposits of pus are produced; or the whole may be involved, and the joint become enveloped in a large abscess. In its later stages, the disease extends to the synovial membrane and the cartilages, or recovery may take place, the joint remaining sound.

Pulpy degeneration of the synovial membrane is a very curious and peculiar disease, with the exact nature of which we are yet scarcely acquainted. It was first described by Sir B. Brodie, as a morbid alteration of structure peculiar to the articular lining membranes, nothing analogous having been observed in the serous sacs. He says: "The disease seems to commence in the reflected portions of the synovial membrane, converting them into a light brown, pulpy substance, varying from a quarter to a half, or even a whole inch in thickness, intersected with white membranous lines and red spots, formed by small vessels injected with their own blood. It then attacks the synovial membrane of 'the cartilages,' beginning at their edge and extending gradually over them, ulceration in those cartilages going on correspondently, till the carious or ulcerating surfaces of the bone are exposed. The cavity of the joint sometimes contains pale-yellow fluid in the floating flakes of lymph, or pus, which is discharged externally by ulceration; but sometimes neither. Or abscesses may exist in the altered synovial membrane itself, without communication in the joint." We have given, in the Pathological Report for 1848–49, a detailed account of the disposition and structure of the synovial membrane thus peculiarly altered, from which we extract the following summary: The new growth formed prominent fringes of a soft, grayish structure, which overlapped and encroached considerably on the surface of the articular cartilage. The marginal zone of the cartilage, for a varying extent, was converted into a kind of fibrous tissue, and blended with the altered synovial membrane. More internally the cartilage was grooved on the surface, probably so as to correspond with the overlying fringe. The fibrous tissue into which the cartilage was transformed was of an imperfect kind, not divided into distinct fibres, and not containing any of the natural cells, but strewed over with numerous oil drops and yellowish molecules. The

change in the cartilage was effected by extraordinary enlargement of its cells, which were crowded with an endogenous growth of young cells containing each a small oil drop and much clear fluid. At the margin of the cartilage, which was obliquely truncated, the change was most

Fig. 348.

A

(A) Vertical section of cartilage in process of absorption towards the left, and overlapped by the vascularised pulpy synovial fringe. The edge of the cartilage where it is obliquely truncated is continuous with the fibrous tissue on the left hand.

(B) Healthy cartilage-cells from the right-hand side, more magnified.

(C) Greatly enlarged cartilage-cell, containing young cells.

(D) Loculus, from thickened synovial membrane, filled and surrounded with nuclei.

advanced; in the interior the structure was quite natural. The pulpy synovial tissue consisted principally of well-formed nuclei and granular matter, with which were mingled a few fusiform and circular cells. These elements were contained in an enveloping membrane, very thin, of whitish aspect, and nearly homogeneous texture. There existed scarce any trace of stromal fibres, but a good many large vesicles, or loculi, formed of almost homogeneous walls, and filled with material similar to that which surrounded them. Delicate-walled bloodvessels ramified through the mass, but not in great numbers. It seems very probable that the altered synovial tissue promotes and is concerned in the absorption of the cartilage. The disease almost always occurs before the middle period of life. "In general, it can be traced to no evident cause; but occasionally, it is the consequence of repeated attacks

of inflammation." It rarely occurs elsewhere than in the knee, but has been seen in the ankle, and in a joint of the fingers. Sir B. Brodie classes it with malignant disease, but from this it is differenced by well-marked characters.

Another very curious alteration, which is sometimes observed in the synovial membrane, consists in its free internal surface being covered by a growth of large villous processes, quite perceptible to the naked eye, which hang into the cavity of the joint, and present a shaggy appearance. They are not developed on the surface of the cartilages. They have sometimes the form of simple threads, or flattened shreds, or their free extremities are split into filaments, like a tassel, or they have a club shape, or resemble melon-seeds, hanging singly or in clusters from each stalk. In structure, they consist of a fibroid material, containing, we believe, in many instances, more or less fat, and approaching herein to that peculiar form of fatty tumor which is called *Lipoma arborescens.* The healthy texture of the articulation is not materially interfered with, at least in many cases. They seem to be the result of a slow exudation of plasmatic matter, which may pass into a low form of organization.

Fig. 344.

Fimbriated knee-joint; the surface of the patella is the only part unoccupied.

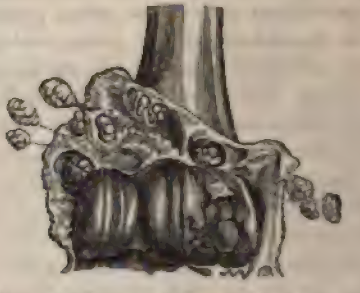
Inflammation of the ligaments, both acute and chronic, is said to occur; but Wickham states that, according to his experience, "the ligaments are the last of all the different parts diseased, and that it is very common to find the ligaments perfect, even when every other texture is either altered or destroyed." From Mr. Key's account, it seems that inflamed ligaments become thickened and more pulpy than natural. The areolar tissue which penetrates among their fibres becomes highly vascular, and is probably concerned in producing the softening and ulceration of their substance, which sometimes takes place.

Relaxation of the ligaments may be the result of long-continued chronic inflammation, or of simple disuse of the limb. In the latter case, it has been known to proceed to such an extent as to allow the head of the femur to slip out of the acetabulum. Frequent and heavy strains may produce a similar effect; Mr. Wickham mentions a case in which the leg was so much bent outward at the knee as to be at nearly right angles with the thigh.

Loose cartilages (so called) are not unfrequent in the cavities of joints. They are usually from the size of a millet-seed to that of a pea, but have been met with as large as a walnut. In shape, they are more or less oval and flattened. Their surface is smooth, as if invested by a serous covering, which they sometimes evidently possess, when they are attached to the synovial membrane by a pedicle of varying length. Formations of this kind commence in the subserous tissue, and as they enlarge gradually, make their way inward towards the cavity of the joint, in which at last they become free by the dissolution of the pedicle. Others are formed by a condensation of fibrinous coagula; "they are distinguished," Rokitansky says, "by their uniform smoothness through-

out, by a delicate albuminous investing membrane, and frequently by their manifest arrangement in concentric laminæ." They never contain any of the characteristic cells of cartilage, and appear to consist solely of compressed fibrillating exudation. Occasionally, they are lodged in

Fig. 345.



Trochlea of humerus; showing formation and connection of loose cartilaginous bodies.

ulcerated cavities of the normal cartilage, which might give rise to the idea that they were truly fragments of this tissue, cut out, as it were, by the process of ulceration. We do not believe, however, that this ever happens. Calcareous matter is sometimes deposited in the substance of these false cartilages, and Mr. Rainey describes true bone lacunæ, similar to those seen in the thin plates of the ethmoid, as existing in their interior.[1]

MORBID CONDITIONS OF CARTILAGE.

It is somewhat doubtful whether a true hypertrophy of cartilage ever takes place; but an apparent hypertrophy is not unfrequently observed. The thickness may be increased to treble of that which is normal: at the same time, the tissue becomes very soft and yielding, and shows a decided tendency to break up into fibres, which are arranged vertically to the surface. There is some evidence to show that, at a later period, cartilages, so altered, would waste and disappear. In advanced age, articular cartilages become considerably thinned—at least this is the case in the hip, and, probably, more or less in all joints that are exposed to pressure. Sometimes the cartilage is simply atrophied; in other cases, it is replaced by a semi-translucent, and in others again, by a white fibroid tissue. Sometimes the cartilage seems itself to ossify, being converted into what is called the ivory or porcellaneous deposit. This is a peculiarly dense kind of bone; its Haversian canals being filled up by the earthy salts. Besides occurring as a gradual, almost unperceived change in the aged, it is also met with very constantly in the disease termed *chronic rheumatic arthritis*, of which we shall presently speak.

[1] A specimen removed from a knee-joint by Mr. Lane, exhibited, on section, a central cavity containing some fatty matter; round this was a zone of hard calcareous deposit, so arranged at its inner part as to include cancelli. Lacunæ existed in this zone. The outer zone consisted of a fibroid layer, imbedding very numerous oval and elongated nuclear particles. This specimen was more than usually organized.

The free surface of cartilages is sometimes covered with a thin layer of lithate of soda; and the same matter may also exist in the substance of the cartilage, in the cancelli of the invested bone, and in the subsynovial tissue. It is deposited as the result of gout. It is not uncommon, on opening joints which are not apparently diseased, to find the cartilages more or less deficient at one or more points, and this especially in the parts where they have had to bear the greatest pressure. The cartilage is eroded more or less deeply, so that in the seat of the lesion the bone may be exposed, and this with scarce any traces of inflammation in the synovial membrane. Sir B. Brodie "has many times observed a portion of cartilage of a joint wanting, and in its place, a thin layer of hard, semi-transparent substance, of a gray color, and presenting an irregular granulated substance." This indicates a partial atrophy, and destruction of the cartilage, with imperfect replacement of it by a fibrinous exudation. We have lately examined the knee-joint of a female, æt. 47, who died of pleuro-pneumonia, in which the cartilage of the femoral condyles, and of the patella, was manifestly in a state of chronic atrophy, or "usure," as it has been termed by Cruveilhier. There was a slight injection of one of the natural synovial fringes, but no trace of inflammatory action; the joint, externally, appeared quite healthy, and no complaint had been made respecting it during life. The cartilage of the patella was most affected; it presented, at its external part, an unequal, irregular surface, about the size of a fourpenny piece, which was softened in texture, and roughened by small grayish prominences. The surrounding cartilage was in a commencing state of similar change. In a vertical section of the part most affected, it was seen that the cells near the free margin were enlarged and multiplied, while the matrix at the margin broke up into fibres of various size, quite separate from each other, the larger still imbedding some of the cells. The accompanying cut illustrates this degeneration of cartilage.

Ulceration of cartilage, occurring as an acute or subacute affection, has been much inquired into, and our knowledge respecting it has become tolerably definite and exact. It was formerly much disputed, whether the change was effected by the action of the vessels of the cartilage itself, or those of the synovial membrane, on the incrusted bone. Now, however, we know certainly, that human articular cartilage is entirely devoid of vessels; and we have good reason to believe that those of the surrounding textures are not the effective agents in the ulcerative process. Mr. Key, who contends most for the influence of the vascular fringes, allows that ulceration may occur as a primary affection, independent of the other textures of the joint. If we refer, as we may fairly do, to the instance of the cornea, as a very analogous tissue, we can scarcely hesitate to admit that ulceration is essentially an alteration of the nutrition of the affected texture, and that the influence of the adjoining vessels upon it is only secondary. The perforating ulcer of the stomach is also a striking instance of the truth of this position. It being then admitted that ulceration of cartilage is produced by a special disorder of its own nutrition, we proceed to inquire what has been ascertained respecting the nature and the stages of this diseased action. These were admirably set forth by Mr. Goodsir, in

his well-known paper on the process of ulceration in articular cartilage, an extract from which we subjoin: "If a thin section, at right angles, be made through the articular cartilage of a joint, at any part where it is covered by gelatinous membrane in scrofulous disease, or by false mem-

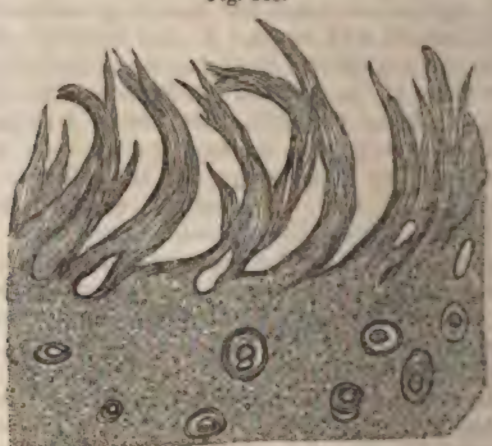
Fig. 346.

Vertical section of cartilage of patella, in state of usure. The free margin presents a number of fibres. The left-hand figure represents one of these fibres, more magnified, and containing some groups of corpuscles.

Fig. 347.

Diseased articular cartilage magnified 240 diameters, showing the enlargement of the corpuscles, the more superficial of which are throwing out their contents into the softened inter-corpuscular substance.—Redfern.

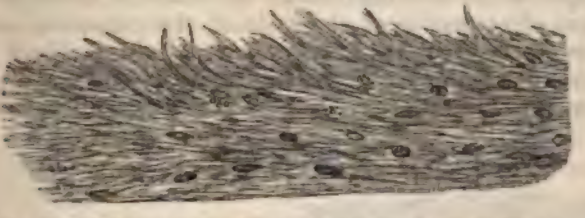
Fig. 348.



Microscopic view of a perpendicular section of articular cartilage, showing its surface occupied by fibrous bands formed by the splitting of the hyaline substance. These bands rendered it velvety in appearance to the naked eye.—Redfern.

brane, in simple inflammatory condition of the joint, and if this section be examined, it will be found to present the following appearances: on one edge of the section is the cartilage unaltered, with its corpuscles natural in position and size. On the opposite edge is the gelatinous or false membrane, both consisting essentially of nucleated particles, inter-

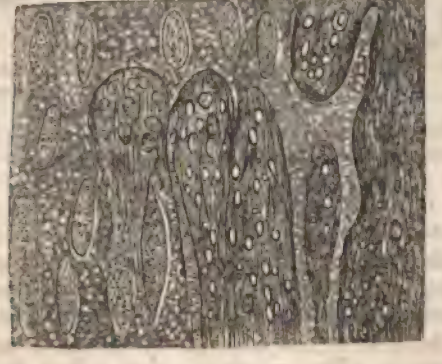
Fig. 349.



Fibrous tissue with included cells and nuclei; formed, as above described, on the surface of the cartilage of the patella.—Redfern.

mixed, especially in the latter, with fibres and bloodvessels; and, in the former, with tubercular granular matter. In the immediate vicinity, and on both sides of the irregular edge of the section of cartilage, where it is connected to the membrane, certain remarkable appearances are seen. These consist, on the side of the cartilage, of a change in the shape and size of the cartilage-corpuscles. Instead of being of their

Fig. 350.

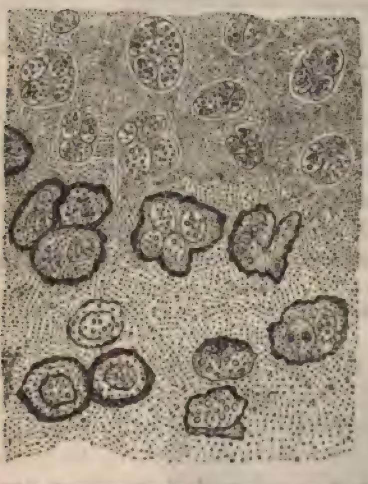


Vertical section of cartilage in a diseased knee-joint, showing the cells enlarged, granular, and bursting. On the right, and above, their contents are seen mingling with a fibrous and granular mass which occupies the surface.—Redfern.

usual form, they are larger, rounded, or oviform; and instead of two or three nucleated cells in their interior, contain a mass of them. At the very edge of the ulcerated cartilage, the cellular contents of the enlarged cartilage-corpuscles communicate with the diseased membrane by openings more or less extended. Some of the ovoidal masses in the enlarged corpuscles may be seen half released from their cavities by the

removal of the cartilage; and others of them may be observed in the substance of the false membrane, close to the cartilage, where they have been left by the entire removal of the cartilage which originally surrounded them. If a portion of the false membrane be gradually torn off the cartilage, the latter will appear rough and honeycombed. Into each depression on its surface, a nipple-like projection of the false membrane penetrates. The cavities of the enlarged corpuscles of the cartilage open on the ulcerated surface by orifices of a size proportional to the extent of absorption of the walls of the corpuscles, and of the free surface of the cartilage. The texture of the cartilage does not exhibit, during the progress of the ulceration, any trace of vascularity. The false membrane is vascular, and loops of capillary vessels dip into

Fig. 351.



Deposition of opaque calcareous matter, commencing in the walls of the cartilage-corpuscles.—Redfern.

Fig. 352.

Drawing of ulceration of cartilage.

(*a*) Vertical section of ulcerating cartilage, magnified.

(*b*) Naked-eye view, showing two ulcerated depressions.

the substance of the nipple-like projections, which fill the depressions on the ulcerated surface of the cartilage; but, with the exception of the enlargement of the corpuscles, and the peculiar development of their contents, no change has occurred in it. A layer of nucleated particles always exists between the loops of capillaries and the ulcerated surface. The cartilage, where it is not covered by the false membrane, is unchanged in structure. The membrane generally adheres with some firmness to the ulcerating surface; in other instances, it is loosely applied to it; but in all, the latter is accurately moulded to the former. In scrofulous disease of the cancellated texture of the heads of bones, or in cases where the joint only is affected, but to the extent of total destruction of the cartilage, over part or the whole of its extent, the latter is, during the progress of the ulceration, attacked from its attached surface. Nipple-shaped processes of vascular cellular texture pass from the bone into the attached surface of the cartilage, the latter undergoing the change already described. The processes from the two

surfaces may thus meet half-way in the substance of the cartilage, or they may press from the attached, and project through a sound portion of the surface of the cartilage, like little vascular nipples or granulations. The cartilage may be thus riddled, or it may be broken up into scales of varying size and thickness, or it may be undermined for a greater or less extent, or be thrown into the fluid of the cavity of the joint in small detached portions, or it may entirely disappear." Mr. Goodsir believes the cells of new formation, the nucleated particles of the false membrane, to be the immediate agents in the absorption of the cartilage. We have had several opportunities of verifying the above account, and have already mentioned two instances in which we observed a similar change in ulcerating or wasting cartilage. We subjoin a short account of another observation, because it seems to show pretty clearly the non-essentiality of the vascular false membrane, or at least of its apposition to the affected part, to the process of ulceration. A child, æt. 4, died with a vast abscess surrounding the right femur; the hip-joint was sound, but inflammation had extended to the knee, the synovial membrane of which was inflamed, and contained some pus. There was an irregular ulcerated surface, of about the size of a shilling, denuding the bone on the trochlear surface of the femur. One of the synovial folds was much injected; its tissue was thickened by interstitial deposit, and adhering to its margin were some masses of exudation, consisting chiefly of amorpho-granular matter, imbedding numerous non-nucleated pus-like corpuscles. The injection of the synovial fold was very marked; its capillaries were distended, and it presented a strong contrast to other parts of the membrane, which were, however, thickened by and covered with exudation. The layer of exudation adhering to the edge of this fold was partially vascularized; but it did not appear that the vessels were continuations of those of the synovial membrane. There was no adhesion between the ulcerated portion of the cartilage and the vascularized false membrane, nor did the two seem to have been in apposition. Near the part where the bone was exposed, there was a small spot of ulceration of the cartilage in progress; it showed to the naked eye two well-marked shallow depressions, and an intervening elevation, with a thin investing gelatinous layer, v. *b*. Fig. 353. In the deeper and healthy parts of the cartilage, as seen in a vertical section, the cells were small and elongated, containing one or more oil-molecules, with faint granulous matter and a clear fluid. Nearer the surface they were a little larger, and had the same contents. The matrix in both these parts was natural, and moderately opaque. At some distance from the free surface, the matrix became suddenly much more transparent, of a pale, homogeneous aspect. The cells in this part were swollen, and of a round shape, appearing to be distended by a clear fluid, in which floated some oil-molecules. They were not much more numerous, except on the ulcerating border, which was pretty even, and covered with escaped cells. There was a good deal of oil, in the form of large and small drops, scattered about; but this did not seem to proceed from the cells or the matrix of the cartilage, in neither of which was there much oil visible; the latter, in particular, appeared simply to liquefy. The investing gelatinous layer consisted of coarse

granular matter, imbedding escaped cartilage-cells and corpuscles like those of pus. The bone subjacent to the cartilage was of a reddish color, was somewhat softened, and its cancelli contained, besides fat-cells, multitudes of granular cells and nuclei. Dr. Redfern has arrived at the following conclusions from his able inquiries into the subject of ulceration of cartilage: "Ulceration in articular cartilage differs from that in other tissues, in neither being accompanied by exudation, nor attended with pain; differences which depend on the absence of vessels and nerves. Ulcers in articular cartilages heal by transformation of the surrounding cartilage-tissue into fibre; but those occurring in other textures are cured by the formation of a cicatrix out of newly-exuded blood-plasma."

SCROFULOUS DISEASE OF THE JOINTS.

No doubt can exist of the propriety of the distinctive name given to this affection, although its course does not seem to be exactly similar to that of scrofulous disease of other parts. The articular extremities of the bones are the primary seat of mischief; they become preternaturally vascular and much softened, so that they are easily cut with a knife, while "a transparent and afterwards a yellow cheesy substance is deposited in their cancelli." From the observation above mentioned, we are inclined to think that granular cells, formed in the primary exudation within the cancelli, play some part in the absorption and removal of the earthy salts of the bone. As the disease of the bone advances, ulceration of the cartilage commences on its attached surface in the manner described in the extract we have given from Mr. Goodsir's paper. Before, however, this can take place to any great extent the articular lamina, so well investigated by Mr. Birkett, must be removed. This consists of a thin lamina of dense bone containing large lacunæ with scarce any canaliculi, which bounds and closes in the cancelli on the surface incrusted by the cartilage. Until this is removed, no vessels can shoot into the nipple-shaped processes of false membrane which dip into the cartilage. The osseous tissue gradually wastes and is absorbed, it undergoes a true caries; sometimes also a part dies and may exfoliate. In the fifty-first case related by Sir B. Brodie there was a small portion of dead bone thrown off into the cavity of the left elbow, while in the right knee, though it presented no manifest indications of disease, and admitted of perfect motion, the bones were unnaturally vascular and softened; and an irregular cavity, occupied by little more than medulla and a reddish fluid, had been formed in the mid-part of the lower extremity of the femur. In cases of this kind, it is not at all unfrequent to find several joints affected with the same morbid change in various stages. As the whole of the articulating surface is generally involved in the disease, the attachment of the cartilage becomes loosened at all points, and it is therefore, even at an early period, much more easily detached from the bone than is natural. Sometimes, as Sir B. Brodie mentions, in the advanced steps of the disease, nearly the whole of the cartilage is found forming an exfoliation instead of being ulcerated. "As the caries of

the bones advances, inflammation takes place of the cellular membrane external to the joint. Serum and afterwards coagulated lymph is effused; and hence arises a pulpy and elastic swelling in the early, and an œdematous swelling in the advanced, stage of the disease. Abscess having formed in the joints, it makes its way by ulceration through the ligaments and the synovial membrane, and afterwards bursts externally, having caused the formation of numerous and circuitous sinuses in the neighboring soft parts." This disease of the joint especially affects children; it is the essence of the *morbus coxarius* which is so very common among the offspring of the poorer classes. It rarely occurs after the age of thirty. Sir B. Brodie cautions us against supposing that all instances of increased vascularity and softening of bone are of the same nature as the disease just described. Simple caries, resulting from inflammation, may produce the same effect, and it is only the deposition of yellow cheesy matter within the cancelli which can be regarded as truly distinctive. The youth of the patient, the simultaneous affection of several joints, and the existence of scrofulous disease in other parts, are also circumstances which will aid us much in forming a correct judgment. M. Lebert, however, denies that the yellow cheesy matter is anything more than concrete pus; he has rarely found any tuberculous matter in bone undergoing caries or necrosis, even in cases of disease of the vertebræ with tuberculosis of several organs. We doubt whether in this M. Lebert has not been led into error, by attaching too much importance to his so-called tubercle corpuscles. Scrofulous matter may contain corpuscles, much more developed than those of tubercle, and yet there will be little difference in the nature and behavior of the two products. Rokitansky and Mr. Paget recognize both circumscribed and infiltrating deposits of tuberculous matter in bone.

Fig. 353.

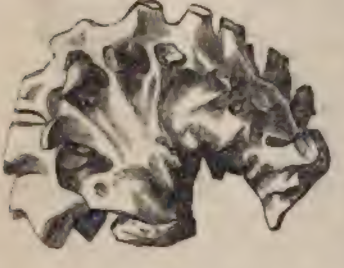
Destruction of cartilage in the knee-joint.—Liston.

DISEASE OF THE SPINAL COLUMN.

The joints of the vertebræ are very liable to be affected in nearly the same way as other articulations of more perfect development, so that a brief account of their morbid states will properly follow here. The cancellous tissue may be the seat of the scrofulous disease just described, occasioning caries and deposition of cheesy matter in the cavities. "In these cases ulceration may begin on any part of the surface, or even in the centre of the bone; but in general the first effects of it are perceptible where the intervertebral cartilage is connected with it, and in the intervertebral cartilage itself. In other cases the vertebræ retain their natural texture and hardness, and the first indication of the disease is ulceration of one or more of the intervert[illegible] cartilages, and of the

surfaces of bone with which they are connected." These cases may be considered analogous to those of primary ulceration of the cartilages of diarthrodial joints. "There is still another order of cases, but these are of more rare occurrence, in which the bodies of the vertebræ are affected with chronic inflammation, of which ulceration of the intervertebral cartilages is the consequence." To this we have not any analogue among the more ordinary diseases of joints. "In whichever of these ways the disease begins, if not checked in its progress, it proceeds to the destruction of the bodies of the vertebræ and intervertebral cartilages, leaving the posterior parts of the vertebræ unaffected by it; the necessary consequence of which is an incurvation of the spine forward, and a projection of the spinous processes posteriorly." The chronic inflammation of the bones sometimes extends to the membranes of the

Fig. 354.



Remarkable example of angular curvature and anchylosis. From a preparation in my museum.—Pirrie.

Fig. 355.

Angular curvature from caries. From a preparation in my museum.—Pirrie.

spinal cord, and when the curvature is very great the cord may be so compressed that it cannot properly discharge its functions. "Suppuration sometimes takes place at a very early period; at other times not until the disease has made considerable progress. The soft parts in the neighborhood of the abscess become thickened and consolidated, forming a thick capsule, in which the abscess is sometimes retained for several successive years; but from which it ultimately makes its way to the surface, presenting itself in one or another situation, according to circumstances. In the advanced stages of the disease, new bone is often deposited in irregular masses on the surface of the bodies of the neighboring vertebræ; and where recovery takes place, the carious surface of the vertebræ above coming in contact with that of the vertebræ below, they become united with each other, at first by soft substance, afterwards by bony anchylosis. This disposition to anchylosis is not the same under all circumstances; it is much less where the bones are affected by scrofula than where they retain their natural texture and hardness; and this explains wherefore, in the former class of cases, a

cure is effected with more difficulty than in the latter. Occasionally, portions of the ulcerated or carious bone lose their vitality, and having become detached are found lying loose in the cavity of the abscess." When a large abscess has formed, the pressure of the matter on the surfaces of the contiguous vertebræ may cause an extensive caries far beyond the limits of the original disease.

ANCHYLOSIS.

The term is derived from the Greek αγκυλη, which signifies a curve of a joint, and was applied to such cases of stiff joint as remained fixed in a curved, not a straight position, which was distinguished by the term ορθοκωλον. The process which produces anchylosis is very similar to that which unites the two ends of a fractured bone, and the union in both cases may be effected either by soft fibroid tissue or by actual bone. For the production of anchylosis it is essential that the cartilage encrusting the articular bony surfaces should be wholly, or in great part, removed. When this is effected, and the conditions of the inflamed parts are such as do not tend to the production of pus, but of plastic exudation, the capillaries, which have entered the processes of false membrane from the bony surfaces, meet and anastomose together; so that the vascular systems of the two bones are in free communication, while the intervening fibrinous exudation gradually undergoes change into a dense fibroid tissue, mingled, as Rokitansky says, with an abundance of fat. If the process stop at this point, soft or fibrous anchylosis is the result. More commonly, however, under favorable conditions of perfect rest, proper food, and attention to the health, the exudation undergoes change into osseous substance, so that the two bones become, as it were, accurately welded together. Mr. South quotes the following interesting instance from Mayo, illustrating what we have described as the early stage of the anchylosing process: "A young man had a lacerated wound of the ankle, erysipelas came on, and matter formed around, and made its way into the joint, the cartilage of which became rapidly absorbed. The limb was removed two months after the accident. Upon a vertical section being made of the ankle, one common change was found to have taken place in both the joints, which the upper and under surfaces of the astragalus contributed to form. In each of these joints the cartilage had entirely disappeared; and the denuded ends of the bones were joined together by a layer of semi-transparent and organized lymph, from a sixth to a quarter of an inch in thickness. This union by lymph was a step towards union by bone. One circumstance appeared to me of peculiar interest. The interior of the bones was perfectly healthy; but the surfaces to which the lymph adhered were, for the depth of one or two lines, extremely vascular." There is a kind of anchylosis which is denominated the *spurious* or *false*, most common after synovitis, and which depends on the presence of masses of exudation within the synovial capsule, with thickening of this membrane and of the ligaments. The condition of the muscles also seems to promote the fixed state of the articulation, the extensors being para-

lyzed and wasted, and the flexors, which exert in most instances a superior power, being contracted, shortened, and atrophied. Anchylosis of the bodies of the vertebræ has already been alluded to as the mode in which a cure takes place after caries of their structure and ulceration of the intervertebral ligaments. It is clear from this that when the destruction of these parts has been at all extensive, the avoidance of the deformity of angular curvature is impossible. Anchylosis of some of the less important joints occurs almost naturally in old age. Some rare instances are recorded in which all the joints of the body became spontaneously anchylosed.

CHRONIC RHEUMATIC ARTHRITIS.

The chronic inflammatory nature of this affection is extremely well marked, but the essential dependence of it upon rheumatism is not so well demonstrated. It occurs not only after an attack of acute rheumatism, but after injuries and bruises, and sometimes without apparent cause. It is very frequent in the hip, the shoulder, the knee, and the articulations of the hand. When it is fully established in the hip-joint, it is said, by Mr. R. Adams, rarely or never to extend itself to the other articulations. Sometimes both hips only are attacked. When the knee is the seat of the disease, or the shoulder, other joints will, generally, be found more or less implicated. In the case of the knee, Mr. R. Adams recognizes a first stage, "marked by evidences of subacute inflammation, such as pain, heat, considerable swelling. This is followed by a second period, in which the heat and swelling diminish, but the pain continues." The disease in the hip and shoulder is described by the same author as of a more chronic character from the commencement, not being attended with any sensation of increased heat, or appearance of distension. A very marked diagnostic sign of this affection is, that pressing the articular surfaces together, and moving them, so as to produce crepitus, does not cause any uneasiness. A similar attempt in ulceration of the cartilages, or in articular caries, would cause severe pain. We quote, from Mr. W. Adams's communication to the Pathological Society, the following account of the appearances ordinarily observed in the advanced stages of chronic rheumatic arthritis:—

"*In the hip-joint.*—1st. Great enlargement and irregularity of shape of the head of the femur, which assumes a mushroom-like form, in consequence of real or apparent flattening of its upper part, and nodulated masses and flattened ring-like layers of new bone, surrounding the edge of its articular cartilage, and extending to a variable distance over its articular surface. To this mushroom-like form, the apparent shortening of the neck, in consequence of its upper part being concealed by the overhanging margin of new bone at the edge of the articular cartilage, also contributes. 2dly. Absence of articular cartilage to a greater or less extent, and the eburnation of the bony surface. 3dly. Nodulated masses of new bone, from the size of a hemp-seed to that of a walnut, attached by thin peduncles to the synovial membrane on the neck of the bone, or to that of the capsular ligament—more or less spherical

when small, but flattened and irregular when of large size. *In the os innominatum.*—1st. Increased capacity of acetabulum. 2dly. Ossification of the fibro-cartilaginous rim, or cotyloid ligament. 3dly. Absence of articular cartilage to a greater or less extent, and eburnation of the exposed bony surface. 4thly. Irregular osseous growths (stalactitic osteophytes) on the surface of the bones external to, and immediately surrounding the joint. *In the knee-joint*, the appearances were essentially similar to those in the hip; new osseous growths, of irregular form, surrounded the margins of the articular cartilages of the femur and tibia; and pedunculated osseous growths, in considerable numbers, and of all sizes, were attached to the synovial membrane, both in the notch and lining the capsule. In addition, however, the articular cartilages on the condyles of the femur presented a thickened, nodulated appearance, in their central parts." Mr. R. Adams, describing the condition of the shoulder-joint, says: "The capsular ligament is occasionally increased in thickness, and its fibres are hypertrophied; and it is generally more capacious than natural, showing that effusion of synovia to a considerable amount had existed, although the external signs of this phenomenon are not usually evident. When the interior of the synovial sac is examined, it will be found to present evidences of having been the seat of chronic inflammation. Bunches of long organized fringes hang into the interior of the synovial sac; and many of these vascular fimbriæ, which in the recent state are of an extremely red color, surround the corona of the head of the humerus. We also notice rounded cartilaginous productions, appended by means of membranous threads attached to the interior of the various structures which compose the joint." The size and shape of these bodies is various. The long tendon of the biceps muscle is very commonly adherent to the superior extremity of the bicipital groove, while that portion of it which normally passes upwards, and takes its attachment to the upper margin of the glenoid cavity, is destroyed. The articular surface of the humerus is very much enlarged, and extends itself over the greater and lesser tuberosities, and even over the highest part of the bicipital groove. The head appears to be in a line with the shaft of the bone, instead of being directed upwards, inwards, and backwards. The cartilage is more or less completely removed, the bone in some parts eburnated, in others porous. Nodules of bone, vegetations, as Mr. R. Adams terms them, are thrown out around the margin of the head. The glenoid cavity of the scapula becomes much enlarged, and, losing its oval shape, assumes a more circular form. This, however, depends much on the position which the head of the humerus occupies. The depth of the articular cavity is increased by osseous productions thrown out around its margins; its encrusting cartilage is removed, and the surface in part is covered by porcellaneous deposit, in part remains porous. The enlarged head of the humerus comes into immediate contact, in many cases, with the under surface of the coraco-acromial vault, causing absorption and wasting of the tendons of the supra-spinatus and biceps, and the upper part of the capsular ligament. The acromion process, the outer extremity of the clavicle, and the coracoid process, in most cases become enlarged, though their under surfaces are worn and ebur-

nated by the friction and pressure of the head of the humerus. Occasionally, however, they are found atrophied, or altogether removed. It is a remarkable circumstance, particularly noticed by Mr. R. Adams—from whose article, in the *Cyclop. of Anat. and Phys.*, we have taken the foregoing account—that, in many cases, the acromion process is traversed in the line of junction of its epiphysis, "by a complete interruption of its continuity, as if fractured." This has been considered by several observers, as well as the destruction of the long tendon of the biceps, to be the result of accidental violence. Cruveilhier is quoted by Mr. R. Adams as describing the bones of the carpus, in a case of chronic rheumatic arthritis of the wrist-joint, to be so confounded together into an irregular mass that it was difficult to say which part each took in the construction of the carpal region. The radius and the ulna undergo like changes to those which have been described above; the lower surface of the latter, confronting the cuneiform bone, becomes smooth and polished, the inter-articular fibro-cartilage having been removed. The nature of the changes taking place in this disease have been admirably investigated by Mr. J. Adams, from whose communication to the Pathological Society, 1850–51, p. 156, we extract the following account. Rokitansky regards the morbid process as an inflammatory rarefaction, attended with swelling and softening of the bone. "After furnishing an osseous exudation within the texture of the bone, and all around, an exudation which may be distinguished by its form and chemical composition, it terminates in consecutive induration." Mr. Adams, from his examinations, arrives at a different conclusion. He believes the process to consist: "1st, in hypertrophy of the articular cartilage, generally occurring at the circumferential margin, but occasionally taking place towards the central parts of the articular surfaces. The new growth of cartilage takes place principally, if not entirely, near to the articular surface." It is very similar, though not quite identical with the original cartilage, a fibrillated character of the matrix, and the scattered, solitary, or imperfectly grouped arrangement of the nuclei being the principal points of difference." 2dly, "in the development of true osseous tissue in the hypertrophied cartilage, ossification commencing either in the newly-formed cartilage or at the junction of the new with the old cartilage. Ossification proceeds more rapidly in the newly-formed and forming cartilage, for its growth is probably simultaneous with the advancing ossification than in the old articular cartilage; so that considerable masses of new bone are formed, altering the configuration of the articular extremities, whilst a layer of articular cartilage remains in its normal position. More slowly, but as perfectly, ossification takes place in this imbedded layer of articular cartilage. The process resembles the normal process of ossification in temporary cartilage in the intercellular matrix being the primary seat of earthy impregnation, and in the enlargement of the cells in the immediate vicinity of the bone."

Effects of Dislocations.—The most common cause of dislocations is a violent strain or injury to the part; but they may also come to pass spontaneously, either from abnormal relaxation of the ligaments, or from destruction of them in consequence of disease and muscular inac-

tion. Dislocation, it is affirmed, may also occur congenitally. What we wish to notice here is, the changes which take place in the articulating surfaces when a dislocation has taken place and remained a long time unreduced. Rokitansky describes these as follows: "The capsule becomes enlarged, and the place of its insertion altered; the articular cavities of the bones increase in size, and undergo various changes in form; and corresponding alterations are produced in the articular heads or prominences. In other cases, in which the dislocation is complete, the capsule wastes, and the bony cavities diminish in size, or are filled with masses of new osseous substance; the displaced head of the bone loses its character, and a new joint is formed. The cellular structures which surround the dislocated head inflame, and frame a new capsule around it, which, for the most part, fits closely, is of fibroid structure, and has a serous lining; whilst the pressure of the head, in its new position, occasions a shallow, articular excavation beneath it. In other cases, instead of an excavation beneath the head, a mass of callus springs up around it, and forms either a hollow to receive it or a level surface, which the head may be flattened in order to fit; or, lastly, the callus may project, and that which was the articular head be excavated to receive it. Sometimes the quantity of new bone deposited around a dislocated head is very abundant, and retains it firmly in its place. In dislocations of long standing, the pressure upon the vessels and nerves interferes with the nutrition of the luxated bone, and, like the soft parts, it is found in a state of atrophy."

Morbid Conditions of Bursæ.—These small synovial sacs are liable to be affected much in the same way as larger. They may be attacked by inflammation, more or less acute, or quite chronic, resulting from rheumatism, the abuse of mercury, or some other constitutional affection, or excited by violence or long-continued pressure. The effusion which takes place may, in cases of a chronic kind, be a simple synovial or serous fluid; but, when the inflammation is more acute, it is either a turbid serum, with flakes of fibrinous matter floating in it, or actual pus. Suppuration sometimes is produced artificially, for the purpose of causing the obliteration of the cavity of the bursa. The matter sometimes makes its way directly to the surface of the skin, and is discharged; but it often, also, escapes into the surrounding cellular tissue, and diffuses itself over a considerable extent. Sir B. Brodie describes this as being of common occurrence after inflammation of the bursa patellæ, so that an abscess is formed between the skin and the fascia," covering the whole of the anterior part of the knee, and liable to be confounded with inflammation of the synovial membrane of the joint. When severe inflammation supervenes, after the puncture of a large bursa, so much constitutional disturbance is sometimes occasioned that the patient dies. This is more likely to occur in persons who are in a state of bad health. The walls of an inflamed bursa sometimes become prodigiously thickened by the organization of layers of fibrinous effusion. There is a specimen in the Museum of St. George's, in which the walls of an enlarged bursa patellæ are more than half an inch thick, while the cavity, which is comparatively small, is traversed by reticulating laminæ of false membrane. "When the inflammation has been of long standing," Sir B. Brodie says,

"it is not unusual to find, floating in the fluid of the bursa, a number of loose bodies, of a flattened oval form, of a light-brown color, with smooth surfaces, resembling small melon-seeds in appearance. There seems to be no doubt that these loose bodies have their origin in the coagulated lymph which was effused in the early stage of the disease; and I have had opportunities, by the examination of several cases, to trace the steps of their gradual formation. At first, the coagulated lymph forms irregular masses, of no determined shape, which afterwards, by the motion and pressure of the contiguous parts, are broken down into smaller portions. These, by degrees, become of a regular form, and assume a firmer consistence, and at last they terminate in the flat oval bodies which have been just described." The synovial sheaths surrounding the flexor tendons of the fingers, as they pass under the annular ligament, are not unfrequently the seat of increased secretion of fluid, and of the formation of small bodies, compared by Mr. R. Adams to grains of boiled rice. "They are found in vast numbers in the same cyst, mixed with a more or less considerable quantity of glairy synovial liquid." They occasion, as they move to and fro, a distinct sensation of *frottement*, and are quite identical with those described by Sir B. Brodie.

Fig. 356.

Enlarged bursa over the patella, the result of pressure. Housemaid's knee.

What are called *ganglions*, are small collections of fluids in bursal cavities of new formation. They are most frequent on the back of the wrist and forearm. They do not seem to arise from inflammation, but rather to be of the nature of simple cysts. They are slightly movable, indolent, and painless, and appear to be situated "in the reticular tissue, which immediately covers the sheath of the extensor tendons." To the latter they are connected firmly, to the skin but loosely. The consistence of their contents varies from that of limpidity to that of thick jelly-like matter.[1] According to Velpeau, these cysts occasionally communicate with the articular synovial cavity.

[1] Some fluid of this kind, which we examined, was coagulated, in great measure at least by nitric acid; it contained a few nucleated granulous corpuscles.

THE PATHOLOGICAL ANATOMY OF THE OSSEOUS SYSTEM.

CHAPTER XLII.

PRELIMINARY REMARKS.

THE peculiar rigidity of the bones, and the large amount of earthy matter entering into their composition, as well as a certain difficulty of making such close and frequent examinations of them as of other tissues, rendered their pathology a terra incognita, until John Hunter demonstrated the close analogy which exists between morbid changes in the hard and soft textures of the body. While we detect with comparative ease variations in the consistency, vascularity, and structure of the latter, the determination of these points in bone is scarcely ever attempted, unless we have to deal with very manifest lesions, owing to the greater physical difficulty which presents itself; hence our knowledge of the early stages of disease, and of the accompanying changes, is less satisfactory than it might be. Fine sections for microscopic examination necessarily alter the relation of, or destroy, the soft parts that enter into the constitution of bone, and therefore deprive us of one important element in the diagnosis of morbid change. Still, much remains to be done in regard to investigating and describing alterations perceptible to the naked eye, and establishing the links connecting certain bone diseases with certain lesions of the system at large, with which we are already well acquainted. The labor necessary to gain this point is probably greater than will be performed in the dead-house of an ordinary hospital; and we must not expect a full solution of such recondite questions of morbid anatomy until we have established endowed professorships, which may enable the incumbents to devote their energies and time solely to scientific purposes.

We have passed over the consideration of monstrosities in other parts of the frame, or of their diseases during the fœtal period. In our remarks on the deformities occurring in after-life, we shall have occasion to allude to some congenital malformations which are persistent; it would, therefore, be inconsistent to review in detail the intra-uterine anomalies affecting the bone. After birth, the functions of the bones may be said to remain almost dormant for some months; but a process of hardening and consolidation is preparing them for the greater tax

that is to be made upon them when the infant learns to shift for itself. It is at this period that our attention is occasionally called to the state of the bones, from their development not taking place in a ratio with the general evolution of the frame. Instances are recorded of a precocious ossification of parts that normally are only membranous or cartilaginous in infant life; but they are unusual. Thus, the fontanelles may close prematurely, or the epiphyses and shafts of the long bones be united by bone. Derangements are much more frequent in the opposite direction; an arrest of osseous growth being caused by a general defect of nutrition more immediately acting upon the process of ossification, or inducing morbid states which indirectly affect the bones.

PERIOSTEUM.

The intimate anatomical and physiological relation of the periosteum to the subjacent bone renders it advisable first to consider the affections to which it is liable. In many instances, the pathologist would probably find it impossible to determine whether disease has commenced in one structure or the other; the more so, as periosteal morbid action may be followed by similar products as we see arising from primary disease of the bone. Incipient inflammation of the membrane is characterized by a red blush, a humid succulent appearance, and more or less of a serous effusion, causing a slight separation from the bone. The periosteum may, as Lobstein[1] observes, be seen to present this condition in the vicinity of chronic ulcers or of old cicatrices. As the inflammation advances, the connection between the membrane and the bone becomes more lax, and the effusion exhibits a purulent character; or the separation may have been so sudden and extensive, especially in adynamic individuals, that, as Dr. Copland shows, before suppuration has time to supervene, gangrene of the periosteum and necrosis of the bone result. An inflammatory process in the periosteum is also essential to the reproduction of bone after fractures, or other lesions of continuity calling for repair. The membrane is peculiarly obnoxious to syphilis and rheumatism—diseases which are prone to fasten upon the fibrous investment of the bones, and induce various secondary disturbances of a more or less serious character. Both chiefly affect the more superficial parts; the periosteum of the skull, the sternum, and the tibia, being the points most commonly attacked. Syphilitic inflammation, or at least that which occurs in the course of syphilis, whether as a result of the virus or of the mercurial treatment, is apt to occur in numerous detached spots, at which tumefaction, induration, the formation of new osseous matter, and necrosis, present themselves. Mr. Stanley[2] remarks, that the hardness of a syphilitic node does not in itself indicate its composition, as he has found supposed osseous nodes to prove mere indurated periosteum. He also states that the pericranium differs from the fibrous investment of other bones in never becoming ossified. Rheumatic perios-

[1] Anatomie Pathologique, vol. ii. p. 83.
[2] On Diseases of the Bones, p. 346.

titis, like the former, presents chiefly the chronic type; it is more liable to occur in the vicinity of the joints, placed here to induce a peculiar form of bony deposit, to which we shall again refer. Another form of periostitis is that frequently met with in cachectic and scrofulous subjects; it is of a sluggish character, causing greater thickening of the membrane and closer adhesion to the bone, followed by suppuration in the tissue, and underneath or upon it. The small amount of pain and constitutional irritation resulting from a lesion, which, under other circumstances, or in other constitutions, would give rise to violent symptoms, is remarkable; while, in a therapeutic point of view, the curability of even very extensive lesions of this type affords a better ground for a favorable prognosis than we should expect *à priori*. This applies also to the cases in which scrofulous periostitis is followed by exfoliations of the subjacent bone; in these cases, there appears to be generally a coincident formation of new bone, sufficient to prevent not only a loss of strength, but even a deformity. In a practical point of view, we should look upon these local manifestations of the scrofulous cachexia rather as a tendency to concentrate diseased action at a distance from vital organs; they ought not, therefore, to be hastily interfered with, but should be regarded rather as a safety-valve to the system, which may be allowed, and even encouraged to act, until the system itself is sufficiently invigorated to elaborate all morbid action. The rapid evolution of scrofulous affections of internal organs, after the cure of the disease just spoken of, as well as the arrest of the former by a local and superficial eruption of the disease, is of too frequent occurrence to permit a doubt of the influence exerted upon the system by scrofulous periostitis. One of the forms of scrofulous periostitis not unfrequently met with, is that giving rise to the severer forms of panaritium or whitlow; the periosteum of the phalanges being the seat of inflammatory action. The longer the duration of periosteal inflammation in any part of the body, the more likely it is that the subjacent bone will become more or less affected by the process—a circumstance easily explicable by the physiological relation existing between the two. It is probable that a large number of the osteophytic, and other osseous growths which form upon the bones under various circumstances, are more immediately the result of periostitis. The large share which this membrane takes in the regeneration of fractured bones, and in the repair of loss of substance from other causes, as shown by surgical observation and physiological experiments, tends to confirm this view. An examination of the preparations of bones sawn through, contained in pathological museums, further establishes the point. Thus we see in St. George's Museum (prep. A. c. 10, H.), a femur considerably enlarged from inflammation; it is in fact a case of eccentric hypertrophy; but the section exhibits the shaft of the bone, though more compact and denser than normally of the ordinary dimensions, traceable through the deposit which has been derived from the periosteum. This deposit, in its turn, exhibits a cancellous texture adjoining the original surface of the femur, bounded by a compact lamella in contact with the periosteum. Here we could scarcely assume the new osseous matter to have been directly formed by the old bone, because in that case we should expect to see either a more entire fusion between

the old and the new formations, or the latter presenting a more complete identity with the former.

Females are liable to a peculiar form of periostitis, especially after parturition; Mr. Stanley, who has drawn attention to this point, states that it is remarkable on account of the severity of its effects, and on account of its liability to cause an error of diagnosis; it effects the pelvis, and mostly its posterior part; and when it occurs near the hip-joint, its symptoms so much resemble those of disease of the joint, that the two are apt to be confounded. A correct diagnosis is material, as the affection is very amenable to treatment.

Mr. Stanley describes a malignant disease of the periosteum, which he has met with on the bones of the hip, and which he attributes to long-continued and repeated attacks of inflammation, altering its structure, and giving rise to the growth of a fungous excrescence upon the membrane. This is sometimes soft and flocculent on its surface, with a firm, grayish, gelatinous base; at others it consists throughout of a firm gelatinous substance; it is both sensitive and vascular, and appears to possess a considerable tendency to involve the adjacent bone and soft parts; after removal by operation, the disease is apt to recur in the adjoining tissues; it does not, however, appear, from the cases recorded by Mr. Stanley, that secondary formations of a similar character occur in other parts of the body; and he himself mentions one case in which, twenty years after the removal of a leg affected with this disease, the individual continued in perfect health.

BONE.

Authors commonly commence the consideration of the pathology of bone by an investigation of hypertrophy and atrophy—two conditions which are associated with, or are consecutive upon, various primary lesions; they are rather an element, or a symptom of diseased action, than the disease itself. The terms may be objected to, when we find them indiscriminately applied to morbid conditions essentially distinct; thus, hypertrophy is used to designate the increase of osseous matter, resulting from the physiological demands made upon the shaft of a long bone, after it has become curved by rickets, as well as to the numerous forms of bony growths, of a compact or cancellated structure, which we meet with upon the surface of the skeleton. Again, authors use atrophy as a generic term, under which they class diseases so remote in their character from one another, as absorption from pressure of a tumor, and mollities ossium. Under such circumstances, language is rendered rather a source of confusion than a means of intelligence; and it becomes a question whether it would not be advisable to eliminate from special pathology terms which indicate a single element in morbid changes, or rather the result of morbid processes, than the nature of the process. With this disclaimer, we shall first describe inflammatory conditions of bone, and incidentally devote a few remarks to hypertrophy and atrophy, but confine their application solely to an increase or a diminution in the normal size and constituents of the bones.

Occasions are sometimes presented of viewing the various stages of

inflammation in bone; and it is manifest that the osseous textures are subject to an increase in their vascular contents as are other organic tissues. The greater vascularity affects chiefly the lining membrane of the medullary and cancellous portions; the ordinary symptoms of inflammatory action may be noted in their incipient stages in surgical practice, but are scarcely seen in the dead-house, except in conjunction with more advanced disease in adjoining portions of the same bone. The first appreciable inflammatory changes in bone, to use Mr. Goodsir's words, "occur within the Haversian canals; these passages dilate, or become opened up, as may be seen on the surface of an inflamed bone, or better, in a section. The result of this enlargement of the canal is the conversion of the contiguous canals into one cavity, and the consequent removal or absorption of all the osseous texture of the part." Some softening is observed to follow inflammation in its early stages, and this will be accompanied by tumefaction, at first of a more succulent, subsequently of a more indurated character. Acute inflammation rarely takes place, except associated with mechanical injury; the dense structure, and the necessarily slower process of effecting a change here than in the soft tissues, are the reason why disease of the bone commonly presents itself in the chronic form. The results of progressive inflammation are congestion, exudation, suppuration, caries, necrosis, with the coincident, and in many instances, as it were, accidental, increase of bone in adjoining parts. An enlargement of the affected portion is almost invariably met with, and may arise either from the changes which take place in its interior, or by a deposit on its surface, or from both. The specific character of the disease in which the inflammation arises determines whether the compact or spongy parts, the shafts or the epiphyses, of bones are effected, while the part of the skeleton attacked is likewise in a measure dependent upon certain uniform tendencies exhibited by various diseases. The spongy and medullary portions have the greater proclivity to take on inflammatory action, and of the hard bones, those that lie nearest the surface are the most liable to become inflamed. Unless resolution of the first or congestive stage takes place, an exudation of a rose-colored lymph, of a gelatinous appearance, is effected, which as we may observe in the same preparation, passes through a variety of shades, light-red, yellow, greenish, or white, filling up the cancelli, or expanding the Haversian canals. This exudation, in its turn, is absorbed, or becomes organized and converted into new bone, or, yielding to the continued morbid action, a destructive process ensues. In the case of its absorption, or of an arrest of the process, the parts may return to their normal condition, or the bone retains a permanently disorganized condition, which may present either an increased condensation and induration, or an abnormal rarefaction of the

Fig. 357.

Microscopic drawing of inflamed and softened bone.

bone. Of the former, we have a good instance in the minute structure of gouty bone, where, as we are informed by Mr. Ure,[1] the Haversian canals are enlarged, and choked up with cretaceous matter, which also lines the medullary canal; the osseous corpuscles are also found to be larger than usual, rather irregularly scattered, and less distinct, and their canaliculi loaded with chalk, which is shown by analysis to be true carbonate of lime,[2] and not like the tophi of adjoining articulations, phosphate of soda; Mr. Ure states that he has found similar appearances in a femur of a person laboring under rheumatic arthritis. The latter, the rarefied state of bone, is that to which the term of osteoporosis was given by Lobstein. This is a condition[3] commonly met with in rickety individuals, though it is also found in advanced life, in subjects who do not appear to be otherwise liable to rachitis. The affected bone presents an increase of size, and a diminution of density, owing to the tissue being expanded; the surface of the bone is irregular, and very porous. The periosteum, however, is not altered, nor do the surrounding textures exhibit any pathological changes. The medulla may be healthy, or changed in color and consistency. The canals of Havers, and the cells, will be found enlarged according to the seat of the injury, the gradual expansion inducing a thinning of the surrounding osseous layers, and eventually a communication between adjoining cavities. The condition may affect the compact or cancellous tissues alone or together. When the cortical layers are the seat of the change, the appearances may induce the resemblance of caries. Lobstein describes the surface in this case as being covered with a multitude of longitudinal fibres, resembling those of a fœtal skull. He attributes them to the development and the action of the periosteal vessels, which hollow out for themselves channels in the osseous substance. When this variety of cortical rarefaction is raised above the surrounding tissue, it resembles one form of osteophyte, and many of the preparations of caries preserved in museums are referable to the same head. The rarefaction of the osseous tissue which constitutes osteoporosis, though often inducing a considerable increase of bulk, is essentially distinct from the process giving rise to the formation of exostosis, in which the generation of new osseous matter is the characteristic feature; the former being essentially an atrophic, the latter an hypertrophic, condition. The term spina ventosa, though one which has nothing but the prestige of antiquity to recommend it, is applied, among others, to affections belonging to the class just considered. We will not seek to perpetuate it by defining its characters, as it is an arbitrary designation, without any acknowledged and established meaning. Osteoporosis gives rise to fragility of the bones affected with it, and undoubtedly takes its origin in many of the dyscrasiæ which impair nutrition; though we meet with it occasionally in old age, as a mere effect of mal-nutrition, without any well-marked symptoms of a constitutional crasis, or of inflammatory action. An

[1] Lancet, 1847.

[2] In preparing sections of bone it is not unusual to employ putty powder; if some of this insinuates itself in the Haversian canals, we should necessarily find some carbonate of lime, which might mislead the observer.

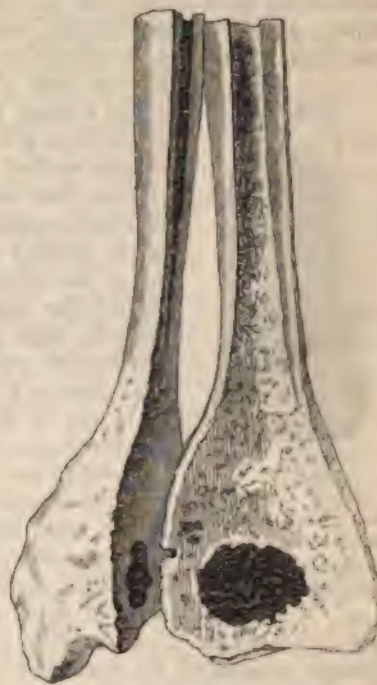
[3] See Lobstein, Anatomie Pathologique, vol. ii. p. 116.

analogy undoubtedly exists between the forms of disease just adverted to and mollities ossium, but the latter presents so peculiar a type, and

Fig. 358.

Suppuration in bone.

Fig. 359.



Abscess in bone.

is so evidently removed from anything like a local affection, or from inflammatory causes, that we reserve its consideration for the sequel, and

Fig. 360.

Abscess in bone.

Fig. 361.

Abscess in bone.

now turn to examine the phenomena of suppuration, and the formation of abscess in bone. This may be diffused, or circumscribed, as in soft parts. It is secreted by the medullary membrane, and in its turn excites more or less reaction in the surrounding textures. The diffused form is the most dangerous, and may arise both from severe mechanical injury, and from constitutional causes. It is commonly associated with phlebitis. The pus occasionally penetrates the bone, and an escape having been thus effected, a cure may be brought about without loss of limb, though the partial death of the bone will necessarily result. In circumscribed abscess we find a cavity, generally in or near the epiphyses, lined with a vascular membrane. A process of condensation or ossification is seen to take place in the vicinity of the abscess, while there is thickening of the adjoining periosteum, and the surrounding cellular tissue. It is probable, as Mr. Stanley remarks, that circumscribed abscess is in some cases attributable to the softening of tubercular matter, analogous to a pulmonary vomica, and that the contents may be discharged, leaving a cavity resembling a tuberculous cavity of the lungs. The circumscribed abscess, to use the words of the same author, usually remains of small size, but in some cases it has enlarged much beyond the natural limits of the bone. Such an enlargement of the abscess is not the effect of simple expansion of the walls of the bone; for in some of these cases the osseous wall of the abscess has increased in thickness with the enlargement of the cavity. The process consists of the combined action of absorption on the inside of the abscess, and of osseous deposit on its outside, whereby its osseous walls may acquire any degree of thickness, according to the predominance of absorption in the one direction, or of deposit in the other.

Fig. 362.

Limited internal abscess in lower part of tibia. Section of bone.—Prepared in Royal College of Surgeons' Museum.

CARIES.

Chronic suppuration accompanies ulceration of bone, or, as it is commonly called, caries, a slow absorptive process, which may occur in all bones, and every part of their structure, but is most liable to attack the cancellous tissue. The bone presents a more or less eroded and cribriform appearance, and while absorption removes the tissue at one point, the surrounding parts are more vascular and tumefied than in the normal state, and the adjoining bone is commonly the seat of new osseous deposit; or the softening process extends to a distance, and the bone is converted into a soft, pliable mass. The more the earthy matter has been absorbed, the fewer the number of osseous corpuscles that remain; until they disappear altogether, and in their place we see only a granular substratum, with faint traces of the lamellar structure. The secretion is of a sanious, acrid, and fetid character, owing to the decomposition which takes place, and which is of diagnostic value during life, in determining the

existence or non-existence of disease of the bone. The discharge also contains minute portions of bony matter, showing the disintegration that is proceeding; and it is very frequently mixed up with a grumous, flaky matter, resembling tubercle, evidencing the scrofulous character of the disease. The discharge is apt to discolor the exposed bone, and itself become of a dirty brown, after exposure to air, as well as to blacken the tissues and probes brought into contact with it. Caries may commence in the interior of a bone, as well as on the surface. In the former case, it commonly makes its way outwards. When the caries is superficial, the Haversian canals are enlarged, and, to use Rokitansky's description, the tissues within them form, in part, a disorganized, soft, and shreddy mass, infiltrated with ichor, or spongy granulations, which easily bleed, grow from them luxuriantly, and advance outwards, over the rough surface of the bone, whilst, internally, they partially or completely fill the enlarged Haversian canals. In both cases, the bone appears porous or cancellous, but its color differs in the two. In the former, it is discolored by the contents of the Haversian canals; in the latter, it obtains various tints of red, from the color of the granulations. When caries affects cancellous tissue, the bone acquires a livid red color, especially if the granulations be at all abundant; it becomes soft, resembles a mass of flesh, traversed by a delicate and brittle bony skeleton, and is easily cut with a knife, or yields to light pressure with a finger; lastly, it becomes swollen. The analogy of caries to ulceration of soft parts, is manifested in the mode in which a cure is established, as well as in its destructive stages. A healthy stimulus being set up, the absorption is converted into a repro-

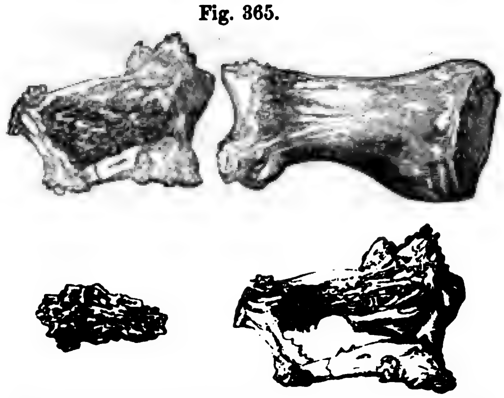
Fig. 363.

Caries.

Fig. 364.

Caries of the elbow; mainly affecting the condyle of the humerus. The vegetative effort around the carious surface well exemplified.

Fig. 365.



Necrosis and caries combined; in phalanges of the toes. In the upper, the carious cavity is represented as still containing its sequestrum. In the lower, the cavity and sequestrum are separate.

Fig. 366.

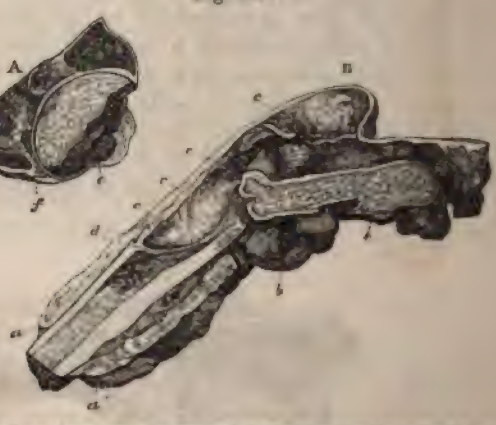
Example of caries in the metatarsal bone of the great toe. Two carious ulcers: each surrounded by interstitial absorption as well as by attempts at reparative effort.

ductive process; the granulations restore the lost parts, if the chasm does not offer too great a tax upon the system, or the cicatrix is formed with more or less loss of the original substance.

NECROSIS.

Though caries often accompanies necrosis, the latter is as distinct from the former, as mortification differs from the ulceration of soft parts. Necrosis consists in the death of a portion, or an entire bone, resulting from various internal or external causes, which destroy its vitality. The necrosed part presents a dead white or waxy hue, or a greater

Fig. 367.



Necrosis of the head of the femur, acetabulum, and shaft, subsequent to amputation. A. Necrosed acetabulum and head, completely anchylosed and broken off at e from the neck. f, Remains of the articular surfaces, closely united. B. Neck, and upper part of the shaft. a, New bone; b, new bone undergoing necrosis. c, bone still containing bloodvessels, and in various stages of inflammation; d, a membranous septum, marking the boundary of the dead and living bone.

intensity of discoloration passing through various shades of green, brown, and black; changes attributed to the influence of the atmosphere, or of the decomposing pus, though not necessarily due to either. The limits of the necrosed portion are not always easily defined, and, at all times, its outline is extremely irregular. It attacks chiefly the compact tissue, and is, therefore, most frequently met with in the shafts of long bones,

from which it rarely extends into the epiphyses, though instances of its doing so are on record, as in the case of the tibia; in the instance from which Fig. 367 is taken, the process is seen to extend through the neck of the femur into the head of the bone, and even to affect the innominatum. The relative frequency of its occurrence in different bones, is stated to be in the following order: the tibia, the femur, the humerus, the cranial bones, the lower jaw, the last phalanx of the finger, the clavicle, ulna, radius, fibula, scapula, upper jaw, pelvic bones, sternum, and ribs. Though a reparative process is commonly set up in the vicinity of the dead bone, which consists partly, in an attempt at separation and elimination; partly, in the reproduction of new osseous matter around the necrosed portion, the effort of nature rarely suffices for this purpose,

Fig. 368.

Fig. 369.

Fig. 370.

Fig. 368. Necrosis of the femur, after amputation. At *a*, the sequestrum in process of separation. At *b*, the parent bone enlarged, and undergoing inflammatory change, necessary for detachment and repair.—Liston.

Fig. 369. The sequestrum detached; at its lower part, *a*, including the whole thickness of the bone. Gradually shelving upwards, as such sequestra usually do.—Liston.

Fig. 370. Sequestrum; seen laterally; the external portions smooth, the internal rough and irregular.—Liston.

and it is necessary that surgical interference should aid in the removal; otherwise, the powers of the individual will probably be exhausted by the continued drain upon the system. A remarkable instanee of the capabilities of an unaided constitution occurred to us, while investigating the effects of the powers of phosphorus in producing necrosis of the maxilla. A man came under our notice, in whom the destructive process had caused death of the body and rami of the lower jaw to such an extent, that the entire bone, with the exception of the condyloid processes, came to lie loose in the cavity of the mouth; a new jaw having, in the

interval, formed underneath, and not, as is usually the case, as a capsule to the necrosed part. The patient's mouth not being of sufficient size for the extraction of the bone, he sawed it across with his own hand, and then extracted it with ease. It is, however, rare for the sequestrum to be so completely separated from the living tissue by the inflammation and suppuration set up in its vicinity, and we even have much difficulty in determining sometimes, in the dead body, the limits of necrosis, as

Fig. 371.

Fig. 372.

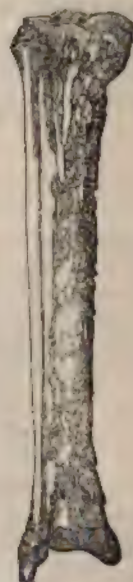


Fig. 373.

Fig. 371. Acute necrosis of the tibia. The bone extensively perished at *a*; the cortical formation has begun to form. Fibula, as usual, unaffected.—Liston.

Fig. 372. Necrosis of tibia, more advanced. Cortical formation investing the greater part of the old bone.—Liston.

Fig. 373. Necrosis of tibia, in the chronic stage. Cortical, or substitute bone complete, and consolidated. At several points cloacæ seen, leading down to the sequestra.—Liston.

its boundaries are imperceptibly lost in the healthy parts. Rokitansky describes the process of separation in the following words: "All round the necrosed portion, that is to say, at its margins and at the part where its surface is exposed to that of the healthy bone, the latter undergoes a gradual expansion, or rarefaction of its tissue, by the enlargement of its Haversian canals, assumes a rosy color, and becomes succulent. It gradually acquires an areolar structure, and is thus more rarefied; at length it disappears altogether, and a red, soft, spongy substance, a layer of granulations, occupies its place. This change is produced by an inflammatory process, which gives rise to suppuration and granulation; the bony tissue, beginning with the Haversian canals, is dissolved by the matter secreted within them, while the granulations which shoot forth at the same time, fill up the enlarged canals. The immediate result of this process, is the formation of a furrow of demar-

cation, which encircles the margin of the dead bone, and is filled up with granulations, and, so far as the process is completed on that surface, also of the living bone, which faces the dead, so far is the sequestrum separated." This process further establishes the analogy existing between necrosis of bone, and the mortification of soft parts, while the description just quoted corroborates John Hunter's opinion, and the investigations of Mr. Goodsir. At the same time, the irregularity of the necrosed portion, and the luxuriant growth of granulations from the healthy part, often cause so close a dovetailing of the two, that it resembles actual organic union, which has misled some observers in their conclusions respecting the nature of the process. Mr. Gulliver has shown, by his experiments, that dead bone may be mechanically introduced into the shafts of a healthy animal, and that a firm adhesion may be established between the two; hence, it is a just inference that, after the morbid process is arrested, and a physiological healing act set up, the sequestrum may be retained as an innocuous foreign body. The experiments of Mr. Gulliver were also undertaken with a view to determining a question long agitated, whether or not bone, once dead, is liable to be reduced by absorption; and his results give a decided negative to this doctrine, which had probably arisen from the circumstance that an exfoliated portion of bone is always smaller than the cavity from which it is removed, owing to the softening and suppuration in the healthy bone, which has allowed of the removal of the former.

Fig. 874.

Necrosis of tibia. At *a*, the dead bone exposed. At *b b*, the papillæ represented, communicating through cloacæ with the sequestrum.

The process of reparation varies according to the seat of the necrosis and the parts implicated.

Necrosis in the outer lamellæ of a bone, when accompanied by destruction of its coverings, including the periosteum, is described by Mr. Stanley as giving rise to the following changes: thickening and consolidation of the inner lamellæ of the bone; inflammation of the surrounding periosteum, occasioning osseous deposit beneath the membrane and in its tissue, and in this way the dead bone becomes circumscribed by a thick projecting border of new osseous substance. By this means, the loss of substance is made to appear even larger than it really is. When the necrosis commences within the bone, a large deposit of osseous matter generally takes place under the periosteum, and in this manner the sequestrum comes to be inclosed in a capsule, which is perforated with openings termed cloacæ; these are not filled up until the dead bone has been removed. After this has taken place, granulations spring from the inner surface of the shell, and the cavity is gradually filled with bony matter; so that, instead of a hollow shaft, we find a solid cylinder. A remarkable fact in connection with cloacæ has been pointed out by Mr. Goodsir, viz: that they are almost invariably opposite a smooth or unaltered portion of the surface of the dead shaft, and that they result

from the pus thrown off from the granulating internal surface of the new shaft making its way to the exterior, by those parts not yet closed, in consequence of having been opposite to portions of the old shaft, which had not afforded separate osseous centres. For those not familiar with Mr. Goodsir's researches, we may add that this fact is one upon which he bases his doctrine, that the reproduction of new bone depends not so much upon the periosteum as upon the spiculæ of bone which remain attached to it, and which act as centres of ossification. "When the entire shaft of a bone," he says, "is attacked with violent inflammation, there is generally time, before death of the bone takes place, for the separation of more or less numerous portions of its surface. When the entire periosteum has separated from the shaft, it carries with it the minute portions of the surface of the bone. Each of these is covered on its external surface by the periosteum; on its internal, by a layer of granulations, the result of the organized matter which originally filled the Haversian canals; the gradual enlargement and subsequent blending of which ultimately allowed their contained vascular contents to combine with the layer of granulations just described, and to form the separating medium between the dead shaft and its minute living remnants. These minute separated portions, after having advanced somewhat in development, appear, when carelessly examined, particularly in dried specimens, to be situated in the substance of the periosteum, and have been adduced by the advocates of the agency of that membrane in forming new bones as evidences of the truth of their opinions."

Fig. 375.

Cloacæ.

Mr. Stanley's views differ from those of the just-named author, inasmuch as he attributes the main ossifying power to the periosteum itself, though he does not deny that a capability for restoring lost bone resides also in the portions of original bone, detached from its surface and remaining attached to the periosteum, in the articular ends of the original bone and the soft tissues around the periosteum, or around the bone, if the periosteum has been destroyed.

The surface of the new bone is at first very irregular and rugged; and, if a section be made, the central cylinder and the external capsule exhibit a well-marked boundary. The more time has elapsed for the curative process to have been established, the more the normal appearance of the surface is restored, and the more completely the separation is obliterated. The period in which the necrosis and the subsequent reproduction take place varies much, both according to the cause of the lesion and the constitution of the individual. The more rapid and complete the death of a portion of bone, the more speedily, on the whole, the reparative process is found to take place; while a sluggish and protracted dying of the osseous tissue, especially when resulting from causes connected with a cachectic state of the blood, or other constitutional maladies, will be followed by a slow process of regeneration.

RACHITIS.

The most important and prevalent disease of mal-nutrition—to employ the most general term that suggests itself—which we have to consider in the osseous system, and which modifies the characters of other maladies that supervene, as it tends to affect the entire bodily frame, is that known as rachitis, or rickets. While frequently associated with inflammatory conditions, it is not essentially an inflammation; and, while one of its symptoms is a diminution of the consistency of the osseous texture, it neither exclusively consists in rarefaction of the bone, nor does it manifest those organic and chemical changes which constitute the peculiar disease known as mollities ossium. Owing to one feature having been regarded by different observers as characteristic of the disease, analogies have been repeatedly set down as proofs of identity. While a general debility is the chief constitutional feature of the disease, a deficiency in the earthy matter of the bones is the chief local phenomenon; yet, as

Fig. 376.

Section of a rachitic tibia, from the King's College Museum.

Fig. 377.

Section of the femur of a rickety child cut with a knife. The shaft consists throughout of cartilaginous and gelatinous substances, intermixed and disposed in cells; it is observable that a greater quantity of cartilage exists in the middle of the shaft, and towards the interior curve, than at any other part. —St. Bartholomew's Museum, i. 34.

Mr. Stanley correctly suggests, the rickety bone is not simply a soft bone, but it undergoes, during the development and subsidence of the disease, a series of curious and somewhat complex changes. The affection is especially one of early childhood; the first symptoms being commonly manifested at the period when we expect the child to leave its mother's arms, and to assert its independence. Guérin found that, of 346 cases, 209 were affected between the first and third years; only three were

congenital; and 34 occurred between the ages of four and twelve; 148 were males, and 198 females. We commonly first perceive a change in the conformation of the lower extremities, which is commonly referred

Fig. 378.

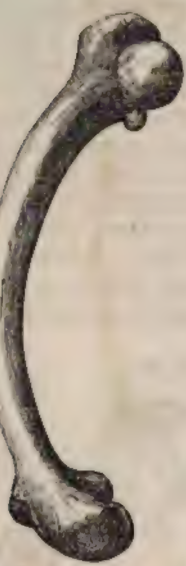


Fig. 379.

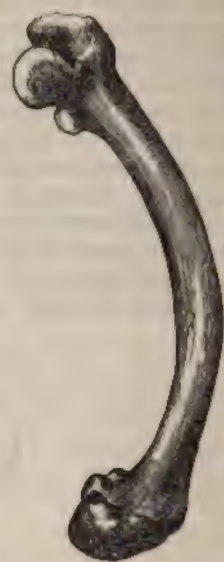
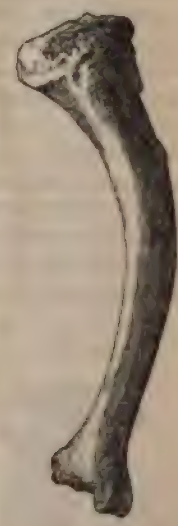


Fig. 380.



Rickets affecting the femur.

Rickets affecting the tibia.

to premature attempts at walking, and the mechanical effect of the pressure of the trunk upon the undeveloped limbs. That physical influences, in some measure, determine the curvature, may not be denied; but that they are but a trifling cause may be inferred from the often surprising rapidity with which, during a continuance of the physical influences, but under improved regimen and medical treatment, the curvatures are rectified. A contortion of the bones of the pelvis, of the spine, the thorax, the upper extremities, and malformations of the skull, may follow upon those of the legs. That the disease does not consist in a simple absence of the due proportion of phosphate of lime, is shown by the coincident tumefaction of the epiphysis, the swelled joints, which but too often add to the deformity just spoken of. The swelling depends upon the exudation of a reddish serum into the enlarged cancelli and canals, the osseous corpuscles, at the same time, exhibiting a deficiency or an entire absence of earthy contents; so that, in cutting a bone thus affected, the knife meets with no resistance. The periosteum, at the same time, is pulpy and thickened, and more than usually adherent to the bone. The chemical constitution of the bone appears to undergo a change beyond the mere absence of the lime-salts; the character of the cartilaginous framework being itself altered in constitution.[1] There is

[1] In deference to the high authority of Mr. Stanley, and on account of the weight which his statements necessarily carry with them, we deem it our duty to advert to an error

a diminution of the salts, varying according to the intensity of the disease; they may be reduced as low as eighteen per cent., while there is

Fig. 381.

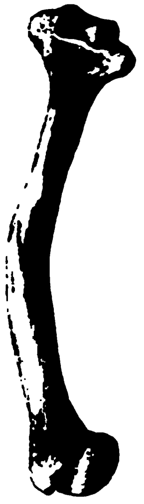
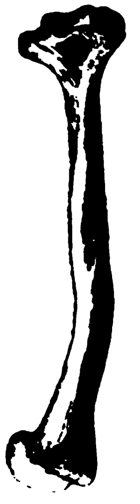


Fig. 382.



Rickets affecting the humerus.

a uniform increase of fatty matter to about six per cent.; it also appears that the fluoride of calcium, of which there is always an appreciable amount in healthy bone, is absent in rickets.

In the analysis of rickety bones, it is important to select the bone before the reparative process is set up. When this is effected, the swellings subside, and the distortions may, by the mere improvement of the general health, be much diminished, and even altogether removed; or, if this be impossible, a deposit of bone takes place in such a manner as to correct the infirmity, and give the patient a limb useful for the ordinary purposes of life. The supplementary ossification is found, on a vertical section of a long bone, to be chiefly on the concave side; so that this part of the shaft may present double and treble the thickness of the opposite side. The structure, at the same time, is very dense, and of ivory texture. In flat bones, as in those of the skull, which is generally unduly large in rickety subjects, there is a uniform thickening, which becomes a matter of serious importance in regard to the intellectual development of the individual, when the cranial cavity is contracted. That this is the case in many cases of idiocy, and especially in that form to which cretinism leads, is very probable. In some instances, the thickening of the bone affects the capacity of the foramina; thus, it has been observed that the foramen ovale has been narrowed

which has crept into his chapter on Rickets (Stanley on the Bones, p. 218). He states that in rickets it has been shown that the cartilage yields neither chondrin nor gelatin; this is based upon an analysis given in Simon's Animal Chemistry (Syd. Soc. Ed. vol. ii. p. 407), of a case of osteo-malacia, and not of rachitis.

by an enlargement of the base of the occipital foramen in epilepsy. In this disease, osteophytic projections are frequently met with, which, however, do not come under the head of rickety productions. A peculiar form of disease of the cranium has been described by Elsässer,[1] as

Fig. 383.

Fig. 384.

Permanent curvature of the spine, with rotation, produced by rickets.

occurring in rachitis, and characterized by softening, thinning, and perforation of the occiput. The bone is atrophied, soft, and porous; and numerous openings are observed along the lambdoidal suture, and in the body of the bone, with the exception of the occipital protuberance. The perforations may amount to as many as thirty; and in place of bone, they are filled up only by the dura mater and pericranium, which are adherent to one another. It is not necessarily a fatal disease; about one-half of the cases are said to prove fatal under symptoms of cerebral and spinal irritation. The affection commonly manifests itself between the third and sixth month of infant life; the child exhibiting much restlessness, and a fear of all contact with the occiput. If it survives, the usual rickety distortions of the skeleton supervene.

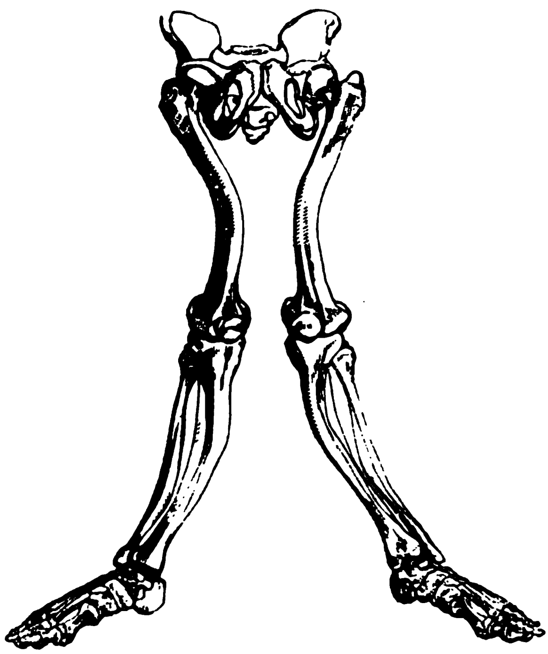
To conclude the subject of rickets, and to avoid repetition, we will at once examine the malformations to which it gives rise in the various parts of the skeleton.

We have already had occasion to allude to curvature of the lower

[1] Der weiche Hinterkopf, ein Beitrag zur Physiologie und Pathologie der ersten Kindheit. Stuttgart, 1843.

extremities; it is one of the most ordinary symptoms of rachitis; it varies in amount from a slight deviation of the bones of the leg from their ordinary shape and direction, to the most extravagant distortion, instances of which are preserved in most anatomical museums. The effect of such irregularity is to diminish the stature of the individual, and to render his gait clumsy; but even if the distortion has been rectified or anticipated, the effect of the constitutional derangement is to stunt the growth. Mr. Stanley expresses himself thus on the subject: "In long bones, the defective growth in length is often such that they are not more than half their natural dimension; but in the direction of their thickness it is not so constant; thus, thigh-bones, a third or even a fourth shorter than natural, are often of their natural thickness. Occasionally, other phenomena are observed in the rickety skeleton; some of its bones are distorted, whilst others are of their natural figure

Fig. 885.



Example of limbs deformed by rickets.—Liston.

and length; but from the failure of growth in the direction of their thickness, are so slender as to present the characters of extreme atrophy. In the lower limbs the weakness of the system, which gives rise to curvatures in the bones, also occasions a yielding of the ligaments of the knee and ankle-joints; hence these distortions of these joints, from the yielding of their ligaments, become part of the phenomena of rickets. And there are instances of such distortions of the knee and ankle-joints, unaccompanied by any bending of the bones.[1]

[1] Stanley on the Bones, p. 224.

The distortions of the pelvis resulting from rachitis constitute a subject deserving the serious attention of the accoucheur; their bearing upon the health and life of the married female ought to stimulate the medical man to have an especial regard to the prevention of pelvic malformation in the female child. A sort of natural protection appears to be afforded to the pelvis against the inroads of rachitis, for numerous instances are recorded in which individuals, with well-developed rickets in the lower extremities and spine, escaped any distortion of the pelvic bones. With few exceptions, the irregularities of the pelvic diameters are due to rickets; they are caused by lateral contraction, by an approximation of the acetabula, by antero-posterior narrowing, from an advance of the sacrum, or by an asymmetrical deformity, due to an arrest in the growth of one-half of the pelvis. In all these cases the mechanism of parturition will be interfered with in proportion to the amount of malformation, and it is the brim which will be found to be chiefly at fault, though each part individually, or all collectively, may be involved in the deformity. "In most cases of partial deformity at the brim," observes Dr. Ramsbotham,[1] "the lateral diameter is increased in size nearly in the same proportion as the conjugate is diminished; but however much the width from ilium to ilium may exceed the ordinary dimensions, the increased space thus obtained will in no degree make amends for the diminution from the sacrum to the pubes; because it is necessary that there should not exist less than a certain quantity of available room in every direction to permit the child's transit." From the sacrum having to support the entire spinal column, the lower lumbar vertebræ and the base of the sacrum are very apt to be thrown forward where there is deficient cohesion, and the consequence will be a diminution of the conjugate diameter. In this case the diameter of the outlet is frequently found enlarged. The ilia will not present the usual expansion, the crests of the ilia will be nearer to one another than in a normal pelvis, and the female will probably also present a hollow-backed appearance. The sacrum is commonly deprived of its concave form, and exhibits a more rectilinear anterior surface, or as Smellie has observed, the vertebræ that compose it ride over one another, and form a protuberance in the part that ought to be concave. These malformations cause those varieties in the form of the pelvis, which have been termed the elliptical, heart, or kidney-shaped, or figure of eight pelves. An oblique form, in which the ilio-pectinal eminence of one side approaches nearer to the promontory on one side than on the other, was first shown by Nagelé to result from anchylosis of one sacro-iliac symphysis; these pelves present a very characteristic appearance, and look as if one half of the pelvis and the acetabulum had been forcibly pushed over to the opposite ilium; hence, the diameter from the sound sacro-iliac union to the opposite acetabulum will be very much diminished, while the interval between the anchylosed symphysis and the other acetabulum will be not only not diminished, but even increased. An excellent delineation of this and several other forms of distorted pelvis are to be found in Dr.

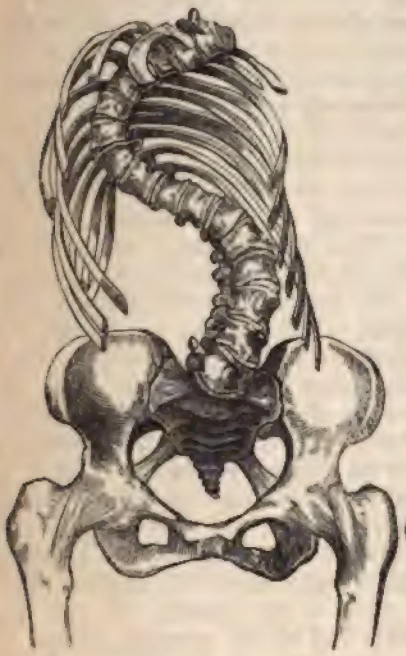
[1] The Principles and Practice of Obstetric Medicine, p. 39.

Ramsbotham's *Atlas of Midwifery*.[1] The rickety distortions of the pelvis are probably never met with unaccompanied by spinal curvature; though the latter may occur without materially influencing the pelvic diameters.

SPINAL CURVATURES.

To one variety of spinal curvatures we have already alluded, that in which there is a projection inwards of the lumbar vertebræ—lordosis. This, however, is not ordinarily a primary affection of the spine, but one secondary to a curvature that has formed higher in the column, and, owing to the sigmoid form of the normal spine, calls for a compensating inclination in the opposite direction, which will necessarily take place

Fig. 386.



Front view of lateral curvature of spine.

Fig. 387.

Back view of same preparation.

where there is a natural tendency forwards. It may also result secondarily from obliquities of the pelvis and coxalgia, which would, however, act differently from the last-named lesion, inasmuch as they would induce a lateral deviation, as well as a projection forwards; in so far, therefore, we differ from Mr. Shaw's views, who states that rickets have no share in producing lateral curvature in females. The main primary curvature of the spine belonging to rachitis, and the one that is more im-

[1] The Principles and Practice of Obstetric Medicine and Surgery, &c., by Francis H. Ramsbotham, M. D., London, 1841.

portant than any other, on account of the frequency of its occurrence, as well as on account of the misery it inflicts upon the patient, and the great deformity produced, is kyphosis, also known as the angular curvature, Pott's malady, or the hump-back. This is almost invariably the result of inflammation and caries of the bodies of one or more of the dorsal vertebræ, or of their intervertebral substances, causing a collapse of several vertebræ, and consequent backward projection of their spines, and an approximation of the corresponding ribs. This disease universally commences early in life; previous to, or about the period of, second dentition. A backward curvature occurs later in life as the result of senile atrophy of the bodies of the vertebræ, which has nothing in common with the angular curvature of rachitis. Lateral curvature of the spine, or scholiosis, is rarely of a rickety character—it may be primary or secondary; and presents an illustration of the law of compensation, equally with the curvatures previously considered. To avoid returning to the subject of spinal curvatures, we add the following remarks on this deformity. The primary curve generally occurs in the dorsal, while the compensating curvature, in the opposite direction, is found in the lumbar region; and as the former is most frequently to the right, the latter, as a legitimate consequence, is most often to the left side. This distortion chiefly affects the female sex, and may be brought on by irregular muscular contraction, or by deficient action of the muscles of one side of the trunk, whether owing to want of exercise, or actual disease, such as pleurisy, or a paralytic affection. When the deformity results from rickets, the primary curvature will probably be found to have taken place in the lumbar region, while the dorsal is secondarily affected. The various malformations of the spine, which we have considered, are not always isolated; but may be complicated with one another, as they are associated with deformities of other parts of the skeleton. That the thorax should be implicated whenever the dorsal vertebræ of the spinal column are affected, may be inferred from the relation the latter bears to the cavity, as well as to the ribs and the sternum combining to form it.

The most common malformation of the thorax consists in a flattening of the sides, with a projection of the sternum, and a swelling of the sternal ends of the ribs; this gives rise to the so-called pigeon-breast. It is very frequently, but not necessarily, associated with angular curvature of the spine; for, in some cases of this disease, the ribs are raised and not flattened, and the lower end of the sternum, instead of being forced out, is actually drawn in, owing to the ribs not being lengthened, and the thorax, in consequence, assumes a more globular form. The thorax, in all cases of rickety distortion, approaches the pelvis unduly, and the abdominal cavity will thus be diminished. A depression of the sternum is very common in rickety subjects; the whole length of the bone being marked by a more or less deep furrow, while the ribs are curved outwards. In both cases just mentioned, the sternum does not deviate from the mesial line; a displacement of this bone, as well as of the thoracic parietes, accompanies lateral curvature of the spine; in this case, to employ the description of Rokitansky, the thorax seems displaced in the opposite direction to the convexity of the dorsal curve, and the whole, or more commonly the lower end only of

the sternum, swerves from the mesial line in the same direction; the axis of the thorax itself inclines towards the convex side of the dorsal curve. One consequence of this deviation is, that that half of the thorax which is on the convex side of the curve is lower than the other, and approaches the pelvis; when there is considerable curvature, the false ribs touch the ilium, or even project into the iliac fossa. But, in extreme cases of combined lateral and posterior curvature in the lower dorsal region, the thorax assumes the contrary position; the ribs which pass from the concavity of the curve, force the chest to the opposite, the convex side; the sternum diverges in the same direction, and the sunken half of the thorax is that on the concave side of the curve. The ribs are packed closer together on the concave than on the convex side; hence the dimensions of the two lateral halves of the thorax are much altered; the one on the concave side being contracted in its antero-posterior, but enlarged in its lateral diameter, while the reverse is the case on the convex side. The ribs, independently of any morbid change of structure, suffer considerable changes in form and outline in these deformities; becoming more or less flattened, and being more or less turned on their axis, according to the dislocation of the vertebræ. The scapulæ follow the distortion of the spine, and also exhibit other evidence of being the actual seat of textural derangement. The upper extremities present similar distortions to those seen in the lower extremities in very advanced cases of rachitis; the bones are ill developed, flattened, and variously curved, while the epiphyses are enlarged. Dr. Farre[1] states that he has met cases in which the upper extremities were bent by rickets, when the lower extremities and the rest of the body exhibited no signs of the disease. If any further proof were required that rickets is essentially a constitutional disease of the same family as scrofula and tubercle, and that its phenomena are not the mere result of mechanical pressure, such cases as those of Dr. Farre would afford it; still, it is important not to overlook the physical influence of the weight of the body in promoting distortions, as we thereby obtain a valuable indication for treatment; for, while everything should be done to correct the vitiated state of blood, it is wise at the same time to remove all unnecessary strain or pressure from any part of the frame, and to afford such support to the weaker points as mechanical ingenuity may suggest.

[1] Quoted by Mr. Stanley, *loc. cit.* p. 226.

CHAPTER XLIII.

ADVENTITIOUS GROWTHS.

UNDER this head we shall consider the various enlargements of an homologous character, termed exostoses and osteophytes, and among which we may also class enchondroma, as well as the heterologous growths met with in bone. Bony tumors are commonly treated of as hypertrophies; we adopt our arrangement partly for convenience, and partly because, as we have already stated, there is a broad distinction between the increase of the normal texture from mere hyper-nutrition, and the grotesque and extravagant forms springing out of various morbid conditions, to which we shall have to advert. Besides, the various forms of so-called hypertrophy are so frequently complicated with other diseased conditions, that it is impossible to determine which group predominates; nor can an arrangement of the tumors of the bone, as Mr. Stanley observes, be founded on the place of their origin, since many of them, identical in nature, arise indifferently from the periosteum, the compact, or the cancellous tissue of bone.

ENCHONDROMA.

We follow the example of Mr. Stanley, and consider, first, the abnormal production of cartilage in connection with bone; or, as it has been termed, by Professor Müller,[1] enchondroma. It consists essentially of the same chemical and microscopic elements as true cartilage, and occurs more frequently in bone than in any other physiological tissue of the body; the bones of the fingers and toes being chiefly liable, though the ribs, vertebræ, and sternum are not exempt; and cases are recorded where the skull, the ilium, and the long bones have been attacked.

Müller refutes the theory of its belonging to the family of scrofula, and attributes it to a peculiar formative process in bone, in consequence of which the embryonic primitive formation of cartilage takes place, and is kept up without the attainment of consolidation, or the more perfect organization of the products. The enchondroma appears to possess an independent vitality; it is radically cured by amputation, and appears never to enter into combination with any other changes in the bone.

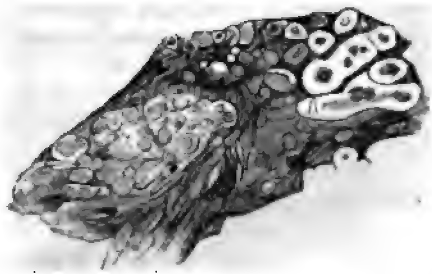
The tumor may originate within the cancellous tissue, or on the surface of the bone. The rapidity and extent of its growth varies, but generally it is of slow progress, and does not exceed the size of an

[1] Ueber den feineren Bau der Krankhaften Geschwülste, 1838, p. 31.

orange. When seated within the bone, the latter gradually expands with the development of the tumor, yet it is unaccompanied by pain or disorganization of the adjoining parts; when external to the bone it exhibits a lobulated arrangement, and is surrounded by a fibrous sheath.

The central variety presents a semi-elastic feel, and, on section, the knife passes through a thin crackling shell of bone, and then exhibits a white cartilaginous mass, which is occasionally found to contain some small cells, while, in some tumors there is an interlacement of fibrous tissue, in which the cartilage is imbedded, thus approximating to fibro-cartilage. They may be solitary, or occur in large numbers in the same

Fig. 888.



Enchondroma. Portion of the tumor removed from the lumbar vertebræ of a soldier, consisting of nodules of cartilage of various forms, with the microscopic features of fœtal cartilage. In the centre of some of the nodules there are small portions of cancellous bone; the centres of others are softened.—St. Bartholomew's Museum, xiv. ii.

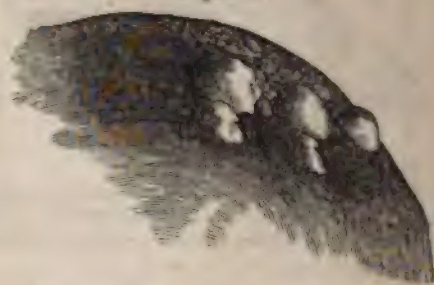
individual. A remarkable instance is recorded in the Reports of the Pathological Society of London,[1] of a boy, in whom the slightest blow produced tumors of this kind. At the time of observation, he presented fifteen or sixteen of these swellings, on the fingers and metacarpal bones, one of which had attained the size of an orange, and required removal, solely on account of its bulk. The superficial variety, though microscopically and chemically identical with the central form, is characterized by the absence of an osseous shell; it is met with chiefly in the pelvis, on the cranium and the ribs. Lebert, who confirms the descriptions and all the details of Muller, gives some cases which fell under his own observation, one of which is particularly interesting, as showing the development of the cartilage,[2] the characters of which were not at once apparent to the naked eye from the highly vascular condition of the tumor. There is, generally, no disposition to ossification, though Rokitansky states that he has observed this metamorphosis in the aggregate variety. The disease is chiefly met with in early life, and appears to be commonly due, as Muller has shown, to mechanical injury interfering with the due development of bone at that period. A case, accompanied by a delineation, in which there was partial ossification, is given in Vogel's *Pathological Anatomy*.[3]

[1] 1848–49, p. 113.
[2] Physiologie Pathologique, tom. ii. p. 212.
[3] Dr. Day's Translation, 1847, p. 582.

EXOSTOSIS.

Osseous growths, consisting of true bone, are divided into exostoses and osteophytes; the difference being marked rather by their form and their cause, than by the etymology of the terms, or their proximate constitution. As no theory is implied, and no false impressions are likely to arise by their employment, the names are more suitable than any other that we might select. Rokitansky defines exostosis as a purely bony mass, set upon a bone, forming with it an organic whole, and, where it is possible, originating or proceeding from the bone; when its development is complete, and often at the beginning of its growth, its texture is homologous with that of its base and point of origin, whether compact or spongy. The former is the most frequent; and it attains a

Fig. 389.



Several ivory exostoses clustered on the os frontis.

hardness which has given rise to the term of Ivory exostosis; while its color is generally whiter than that of the bone from which it springs. Of the density of these exostoses the best proof is that operators are sometimes unable to remove them; in St. George's Hospital Museum we find an exostosis from the orbit which sloughed off on the application of caustic, though Sir Astley Cooper had previously failed in sawing it off; there is another specimen in the same museum about one inch and a half in diameter, which took one hour to remove, and more than one saw was spoiled during the time. The exostosis may be entirely sessile, or it resembles a mushroom in its mode of growth, presenting a constriction at its base, which, though it may penetrate deeply, is so fine as to be imperceptible during life. Rokitansky so absolutely denies the complication of the compact and spongy exostosis, that we must specially refer to an instance which was exhibited at the Pathological Society of London,[1] in 1850, and which was remarkable both on account of its size, and because the base and pedicle were compact, while the remainder was cellular. The surface of these exostoses is smooth, and their outline is commonly a segment of a circle, or of an ellipse; their cause is an idiosyncrasy of the individual, not referable, as far as we can trace, to any definite constitutional taint. Some of the hard exostoses we

[1] See Report for 1850–51, p. 149.

meet with are manifestly mere hypertrophies of the normal prominences of the bone upon which they are seated; thus, we see the tuberosity of the tibia, the styloid process and similar parts, give rise to these formations; an instance occurs in the malum coxæ senile, where an exuberant

Fig. 890.

Spongy exostosis on the femur, with a broad base and pointed processes directed downwards; the section shows a cancellous structure, surrounded by a shell of compact bone. The walls of the femur and the medullary cavity, in the situation of the exostosis, are perfectly sound.—St. Bartholomew's Museum, i. 186.

Fig. 891.

Exostosis of the femur.

development of bone, probably owing to an arthritic process, takes place on the trochanter, and round the neck of the former, which it entirely overhangs. The bones of the skull are the most ordinary site of hard exostosis; it is also seen in the long bones, and in the pelvis, where they may prove an obstacle to parturition. The microscopic appearances are described by Rokitansky as exhibiting a very considerable number of peripheral lamellæ, in which long corpuscles are observed. The Haversian canals are small, and far apart, many of them being surrounded by a distinct and isolated system of lamellæ; large tracts present no corpuscles, while at other spots they are clustered together in dense groups. No new tissue is discoverable in the ivory exostosis.

Spongy exostoses differ from the compact variety, in being composed of cancelli, containing medullary matter, and surrounded by a shell of bone; they vary much more in size and outline than the former; they spring from the cancellous or compact tissue of the bone, and their surface is continuous with that of the latter. In some cases the medullary cavity of the bone is immediately continuous with that of the exostosis,

so that this resembles a diverticulum. The spongy exostosis occurs at all periods of life—when it has attained a certain size it generally remains permanent. Rokitansky describes a process of condensation alternating with one of rarefaction; it is by the latter that he considers the growth of the spongy exostosis outwards to be chiefly affected.

OSTEOPHYTES.

The osteophyte was first characterized by Lobstein as a bony vegetation which grows from the surface of the bone, or encircles the articulations, and offers the most varied forms. It is distinct from exostosis, in not forming well-defined local tumors that are more or less circumscribed. It is not developed between the layers of the compact tissue, and their surface is rough, while the texture appears to differ more from that of the matrix than it does in the exostosis. The osteophyte sometimes bears a close resemblance to certain forms of coral. The osteophyte chiefly affects the more vascular portions of bones, as

Fig. 392.

Osteophytes, occupying the lower end of the femur. The whole exterior of the bones is roughened by the growth of irregular plates and pointed processes of osseous substance. A large canal, formed by ulceration, passes through the bone just above the condyles; around the lower part of each condyle is a broad rim of new bone. From a man æt. 35, with long-standing disease of the bone.—St. Bartholomew's Museum, i. 201.

their articular ends, their rough lines, or, in the skull, the sutural cartilages; because, as Rokitansky remarks, it is generally the product of an inflammatory process in the superficial part of the bone, and in the periosteum; and hence it is very commonly found adjoining and surrounding not only portions which are inflamed, carious, or necrosed, but also spots of bone affected with various other diseases, which, in some stage of their existence, have occasioned a reaction in the tissue of the bone. Thus, we may refer the osteophyte, in an individual case, to simple inflammation, to rheumatic or gouty inflammation, to syphilis or

other causes. Gluge[1] appears to admit one variety of osteophyte only, which he describes under the term of osteophyton gelatinosum, as forming by the ossification of a fluid, gelatinous mass, effused on the surface of the bone; the mass consists of granular cells, which are successively converted into cartilage and bone-corpuscles, which are disposed in rows, or layers, forming lamellæ or spiculæ at right angles to the bone; a reddish jelly-like fluid continues to surround the new bone, which is sometimes discharged in large quantities, and thus induces the erroneous assumption that we have to deal with carcinomatous degeneration.

The diffused and fibro-reticular osteophyte of Lobstein, or what Rokitansky terms the velvety villous osteophyte, forms an osseous layer investing a bone that is otherwise healthy, sometimes removable, sometimes firmly soldered to it; it commonly presents the color of the bone, or it may be discolored; by a lens it is found to present a furrowed surface, or to be composed of minute upright spiculæ; the small channels which separate the osseous ridges being in the direction of the vessels of the periosteum. This variety is a very common accompaniment of inflammatory affections of the bone—it is the one which Rokitansky has observed to occur in females dying shortly after parturition, and has, therefore, called the puerperal osteophyte. The subject has not attracted the notice of English pathologists, but the authority of Rokitansky's name renders some attention to it imperative. The layer of new bone, he says, varies in thickness from a very thin film to half a line, and more; generally occupies the frontal and parietal bones, but is sometimes found covering the whole inner surface of the cranial vault, and scattered in patches over the base of the skull. It exhibits the same mode of development as other forms of ossification; it presents itself in all females who die either of puerperal disease, or from other causes, after the third month of pregnancy, or shortly after parturition. The complete incorporation of the exudation with the old bone causes an increased thickness of the latter, which is rendered more evident by repeated pregnancies. Rokitansky appears to regard the puerperal osteophyte as a uniform accompaniment of pregnancy; he does not seem to refer any of the sympathetic symptoms of this condition to the deposit, nor does he state that it is ever reabsorbed; but he enforces his position by the contrast which the frequency of this growth in women who are pregnant, or have been recently confined, offers with its rarity in other persons. Exudations, he says, are deposited on the vitreous table in both sexes and at all ages, but they are less extensive than the puerperal osteophyte, and are usually confined to the neighborhood of the longitudinal furrow. Rokitansky's second variety of osteophyte is the splintered or laminated form, presenting itself in excrescences and lamellæ several lines in length, of a conical shape, and terminating in a sharp point, which are found chiefly in the neighborhood of the cancellous parts of bone affected with caries.

The next form of osteophyte which we have to consider is that which appears to be mainly the result of gouty and rheumatic affections; it is distinguished by forming excrescences of a warty and stalactitic cha-

[1] Atlas of Pathological Anatomy, Art. Osteophyte. American Edition.

racter, which are developed in the vicinity of joints of persons laboring under those diseases; the articular surfaces may be partially absorbed and present patches of enamel-like deposit, while the new osseous formations are thrown out, as it were, to support the defective mechanism. The osteophyte produced under such circumstances sometimes surrounds the joint and gives rise to a bony anchylosis. The bodies of the vertebræ are frequently found united to one another by osseous vegetations extending over two or more bones, like bridges; they are analogous to the callus uniting fractures, but appear to be the result of some constitutional cause. Similar bony ridges are also observed to form between the ribs.

The botryoidal, or cauliflower osteophyte, is described by Lobstein as a large sessile tumor, which is more or less compact at the base, and becomes spongy towards the surface, sometimes attaining the size of the head of a seven-months' child; it occasionally merely forms a capsule to other heterogeneous matter, such as scirrhous, fibrous, fungous tumors, and the like. Lobstein's general theory of osteophytic growths is, that they consist mainly of an ossification of the tissues surrounding the bone; according to this view, the diffused osteophyte is nothing but ossification of the cellular tissue, uniting the periosteum to the bone; the fibro-reticular osteophyte an ossification of the periosteum itself; the flat and styloid form the ossified tendinous and aponeurotic fibres; while the botryoidal variety, and that causing anchylosis, is attributed to ossification of the inter-muscular cellular tissue; he denies the inflammatory origin of the malady, and sets it down to a morbid hypertrophy; though, in that case, it would be scarcely consistent to speak of ossification of the various tissues; but we should regard it as essentially an outgrowth from the bone itself. This it certainly appears to be in the majority of instances; the original constitution or the morbid taint, rendering the bone a nidus for diseased growth; the character of the latter is determined by its matrix. Lobstein's theory, however, may be held to apply in those rare cases in which we find the osseous growths produced indiscriminately in the soft parts and upon the skeleton; Mr. Stanley[1] relates several instances of this kind, in which bony growths have appeared in various situations and in considerable number, either simultaneously or in quick succession. They generally occur early in life; an arrest in the development of the morbid formation taking place at manhood, there is reason for assuming an hereditary predisposition.

FIBROUS GROWTHS.

Fibrous tissue is developed within or upon the bone, and gives rise to tumors, which, in the former case, are surrounded by an osseous envelop; they present more or less elasticity, according to the density of the inclosed growth; offer a gray, opaque character, and yield gelatin on boiling; they occur chiefly in spongy bones, as the articular ends of long bones in the vertebræ, the upper and lower jaw, the scapula and

[1] On the Diseases of Bones, p. 212.

ossa innominata. Fibrous tumors may attain an enormous size: thus, a specimen in St. Bartholomew's Hospital, alluded to by Mr. Stanley, which grew from the humerus, measured three feet in circumference. Though non-malignant, there seems to be a tendency to reproduction after surgical removal, which has not been observed in enchondroma; in which growth we noticed the occasional development of fibrous tissue, and the perfect security from relapse. Fibrous tumors and enchondroma present no features by which they may be distinguished during life; the lobulated surface is not peculiar to the latter. Mr. Adams[1] exhibited a fibrous epuli springing from the cancelli of the lower jaw, of the size of an orange, forming a large lobulated mass.

OSTEOID TUMOR.

A transition form of osseous disease which intervenes between the simple bony tumors hitherto considered, and the malignant affections of bone, is the osteoid tumor of Professor Müller; or, as it is called by Mr. Stanley, the malignant osseous tumor. It has been alluded to by other authors under various terms; but their introduction would only perpetuate the confusion which in pathology cannot be too much deprecated. Müller describes it thus: The osteoid tumor is irregularly lobulated, and is developed with more or less rapidity from the surface of a bone, and consists mainly of osseous tissue, in the interstices of which a non-ossified substance is found of the consistency of fibro-cartilage, which also forms the covering of these growths; the bony matter is more or less porous, and presents all the characters of true bone, while the other constituent offers a grayish-white color, is somewhat vascular, and of firm consistency, and difficult to tear. The microscope displays in it a dense, fibrous network, with minute interstices, containing but few cells and nuclei; it is not cartilaginous in structure or chemical composition, containing neither gelatin nor chondrin. These tumors result from a constitutional diathesis (an osteo-plastic diathesis, as it is termed by Lebert), leading to a local formation of bone, which, in its turn, is destructive to the system. One tumor commonly appears on a bone, and numerous others subsequently form on different parts of the skeleton, and, at last, the osteoid growths are even developed in the soft tissues, whether the primary tumor have been surgically interfered with or not. Mr. Stanley, who gives three cases of the affection, states the characteristic features to consist in a tendency to grow round the lower part of the femur, just above its condyles, and around the upper part of the tibia, just below its head; in a tendency to assume an oblong, rather than the globular form, which belongs to many other tumors of bone; and in the absorbent glands, when contaminated in this disease, assuming the form of hard, isolated, movable tumors.

[1] Report of Pathol. Society, 1847, p. 114.

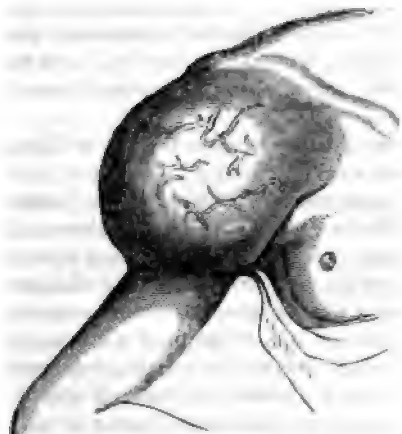
CANCER.

Of the various forms of cancer, the encephaloid variety is found to affect bone most frequently; it occurs either in the infiltrated form, or in the shape of a tumor. Either may be primary or consecutive, though idiopathic cancer of bone is of rare occurrence; it is particularly liable to follow mammary carcinoma. Tuberiform cancer, according to Cruveilhier, differs from infiltrated cancer as lobular from ordinary pneumonia; uncircumscribed cancer being more commonly limited to one bone, or part of a bone, while carcinomatous tumors are commonly the result of a cancerous cachexia. Cancer, in blocks, according to the same author, exclusively attacks the medullary tissue; infiltrated cancer sometimes affects exclusively the periosteum, the bone, or the medulla, or two or more at once; the same individual may present each variety of site, showing the identity of the different forms.

Cruveilhier considers the compact tissue of bones not in itself susceptible of becoming the seat of tuberiform cancer; in the few cases in which it has been found affected, he explains the process by a previous transformation of the compact into spongy bone, a notion regarded by Dr. Walshe as fanciful. In the infiltrated form of encephaloid, the cancelli and Haversian canals are filled with a reddish, fatty-looking substance, which causes an absorption of the cancellar septa, and thus becomes one of the various morbid conditions to which a great fragility of the bones is attributable. A case of primary medullary cancer of the femur, which has fallen under our notice, and was regarded, during life, as a rheumatic affection of the hip-joint, exhibited at the post mortem a remarkable fragility of the affected femur, the neck of which was broken during removal, the cancellar tissue of the bone appeared rarefied and filled with a reddish fat, and it was only by the microscope that the carcinomatous nature of the deposit, which was peculiarly well marked, was revealed. It is doubtful whether the infiltrated may be converted into the aggregated form; encephaloid tumors, however, in bone, attain a considerable size, distending into a cyst, or, according to Mr. Stanley, in some rare instances, being accompanied by increase of thickness of the bone, which gives to the tumor the character of a solid mass of bone. Cruveilhier remarks that it is characteristic of cancer not to give rise to any new development of bone. The shell occasionally yields and breaks, and, the growth of the cancer being unimpeded, increases with sudden rapidity, while the severe pain is lessened. The tissue presents the characters of medullary cancer; it varies in vascularity; at times, either from the large number of small arteries passing through the growth, or from the vicinity of a large artery, it puts on the character of an erectile or aneurismal tumor; the diagnosis in this case is of great importance in reference to treatment, as an erroneous assumption of the vascular nature of the disease would, as it has done, lead to the application of ligatures, a proceeding necessarily useless. The epiphysis of the long bones, especially of the femur, the tibia, and the humerus, are chiefly liable to be affected with medullary carcinoma; the bones of the head and face, the ribs, sternum, and pelvis, are also subject to be

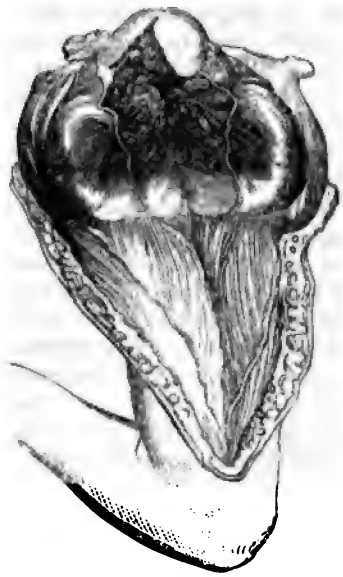
attacked. The fragility which was noticed in the individual case before alluded to is a quality often spoken of by authors as associated with cancer, without determining or assuming the existence of actual carcinomatous disorganization of the bone; it is also observed in deep-seated constitutional affections of a syphilitic, scorbutic, and arthritic character,

Fig. 393.



Osteocephaloma of the head of the humerus, with spontaneous fracture of the shaft.

Fig. 394.



Section of tumor; upper end and head of humerus destroyed, but cartilage of incrustation unaffected. Tumor divided by white vertical lines—the periosteum, inside which only were the osseous spiculæ found.

and cases are recorded of extreme fragility as a mere result of old age; it is not, therefore, inconsistent to assume that, when accompanying carcinomatous affections, it may be the result of an atrophic state resulting from mal-nutrition, without being necessarily accompanied by any actual carcinomatous degeneration of the bone. Lobstein, who devotes an entire chapter to the consideration of fragility of the bones, relates a case of such extreme fragility, in an adult female, that pressure of one finger on the head of the tibia caused the bone to give way: the bones were very porous, and a white, milky fluid exuded from them when compressed; the cartilaginous basis of the bones seemed altogether to have disappeared, and, excepting the glutæus maximus, the abdominal muscles, and deltoid, all the muscular tissue was converted into fat. Rokitansky, who gives a similar case, is of opinion that we have to deal with a peculiar form of encephaloid infiltration characterized by the milky juice.

The areolar or gelatiniform variety of cancer is occasionally met with in bone. Mr. Stanley records a case affecting the bone of a finger, and

another occurring in a rib. Rokitansky relates one in which the right upper maxilla was the seat of the growth, and where the peripheral follicles were developed into large cysts.

Mr. Stanley describes as malignant a peculiar degeneration of the tissue of bone, which appears to commence in the deposit of a yellow substance into the medullary canals, changing its color, and converting its texture into a soft, crumbling, greasy substance; small cells filled with a glairy fluid, and short white brittle fibres, as well as osseous granules and laminæ, are found in it; and as the disease advances, which it does with every feature of malignancy, the morbid deposit extends beyond the limits of the bone in the form of a circumscribed tumor. Subsequently, all the surrounding tissues are involved, the cellular tissue and the adjoining absorbent glands being converted into a similar morbid mass.

Rokitansky describes fibrous cancer as being occasionally met with in bone. A malignant fusion of the bones is described by Lobstein (under the designation of osteo-lyosis) as a rare disease, in which to a greater or less extent the bone deliquesces, leaving in its place a collection of matter of different color and consistency, but not offering any acrimonious character. The existence of the disease is admitted by Rokitansky, who adds that it commences in the diploe, where it forms a cavity, first inclosed between the compact tables; these gradually disappear, leaving an irregular gap, which is covered by the periosteum, destined in its turn to become involved; in this way a bladder is formed, which is filled with gelatinous fluid. It is commonly combined with the development of cancer in the internal organs.

TUBERCLE.

The presence of tubercle in bone is an undoubted pathological fact, but its frequency has been over-estimated by some authors, as it has been underrated by others. In the former case, the error has arisen from every evidence of osseous disease in scrofulous subjects having been regarded as actually resulting from the deposit of tubercular matter in the bone, and from concrete pus having produced appearances closely resembling those presented by tubercular matter. In a question of this kind the microscope must solve the doubt; the high authority of Lebert[1] on pathological microscopy justifies our giving the following extract from his remarks on tuberculization of bone: 1. When the areolar structure of the spongy tissue of bone is yet well preserved, and its meshes filled with pus, which from having no vent becomes concrete, it is apt to resemble yellow cheesy tubercle; when a portion of the fatty matter of the medulla becomes mixed with this cheesy substance, the latter offers a somewhat transparent character, so as to resemble gray granulations; the microscope, however, will detect nothing but the elements of pus and fat. 2. We occasionally meet with abscesses in the middle of a bone, surrounded by a fibrous membrane. If the pus is unable to escape

[1] Physiologie Pathologique, vol. i. p. 473.

by a fistulous passage, it becomes concrete, and then assumes the aspect of what has been described under the name of encysted tubercle of bone. 3. The cavities formed in the vertebræ, in caries of the spine, ordinarily contain nothing but a portion of bone more or less detached, surrounded by sanious pus. These are to be regarded as osseous ulcers, in which we fail to discover the essential element of tubercular caverns, viz: tubercle. 4. It is a common thing to meet with caseous masses occupying pouches in front of the seat of vertebral caries, from which fistulous passages extend and open into the inguinal folds, or elsewhere. These pouches contain a grumous matter, which is nothing but concrete pus mixed with particles of bone. 5. M. Lebert denies not only having found tubercular matter, in those cases of vertebral caries, in which there were no pulmonary tubercles, but states that he has even failed to find it in those cases in which several organs presented tubercular deposit.

Rokitansky's and Nélaton's views differ materially from those expressed by the author just quoted; and it will remain with future inquirers to determine in how far the limitation given by Lebert is correct. Still, the general laws as to the site of osseous tubercle remain the same in either case. It affects chiefly the spongy bones, and the cancellous portions of long bones. An instance of crude tubercle presenting itself in the shaft of a long bone is given by Mr. Hewett in the Pathological Reports,[1] which we quote on account of its extreme rarity. The individual, a man aged thirty, had been laboring, for fifteen months previous to his admission into St. George's Hospital, under a tumor of the middle part of the thigh, supposed at first to be malignant, an opinion which was modified when the swelling was somewhat reduced under treatment. He died eventually of erysipelas—tubercular deposits were found in the peritoneum, kidneys, spleen, and lungs. In the thigh, there was great thickening and condensation about the cellular tissue uniting the muscles, and in that between the muscles and the bone; the periosteum, also much thickened, presented on its free surface a large patch of tubercular matter, enveloped in a dense cyst. The bone itself was irregular in shape, much hypertrophied, and very hard; at this part its medullary cavity was filled with tubercular matter, surrounded by gray semi-transparent lymph, presenting very much the appearance of the well-known granule of the lung.

According to the prevailing views, tubercle commonly occurs in bone, as the yellow opaque tubercle deposited in the cancelli, and inducing a gradual absorption of the bony septa, so as to lead to the formation of larger accumulations; this deposit is liable to softening, by being mixed with the products of inflammation, and is thus converted into a scrofulous abscess, surrounded by what Rokitansky terms a lardaceo-callous cyst, "which," he says, "is in fact the tissue surrounding the softening tubercle, infiltrated with lardaceo-gelatinous material." If the tubercular matter does not soften it becomes cretified; we must attribute the chalky substance often found in the cancelli, to a retrograde process of this kind. We should be disinclined to admit, to the letter, Mr. Stanley's

[1] Report of the Pathological Society, 1850–51, p. 147.

statement, that no reproductive process ever ensues upon the destruction of bone by scrofulous disease; the sluggishness of scrofulous affections of bone is notorious, and undoubtedly in the majority of instances, whether the destructive process affects the shaft of a long bone, or the cancellar structure of a vertebra, the cure is only wrought with a loss of substance giving rise to some deformity. One of the most familiar instances is that variety of spinal curvature known as Pott's malady, in which the scrofulous destruction of one or more bodies of vertebræ induces a projection backwards of their spines, and a shortening of the column. The adjoining soft parts are often extensively involved and secondary effects produced, which do not at first sight appear connected with the original malady; thus in scrofulous caries of the spine, abscesses form in the surrounding cellular and muscular tissues, which may point in the lumbar, sciatic, or inguinal regions, giving rise to lumbar or psoas abscesses, or, in the case of scrofulous disease of the hip-joint, we have necrosis extending to the pelvis, or sinuses burrowing down to the popliteal regions.

VASCULAR TUMORS.

Tumors of a vascular character are occasionally met with in bone, resembling those composed of erectile tissue, or rather of a congeries of bloodvessels, in soft parts. Mr. Stanley describes a tumor of this kind that fell under his observation, as bearing a close resemblance to certain nævi consisting of dilated bloodvessels, with a fibrous tissue occupying their interstices; hence, in a section, the tumor presented a cribriform appearance, the orifices being apparently those of divided bloodvessels. Mr. Stanley regards it simply as a local disease, curable by removal of the affected part; Rokitansky believes that it is of cancerous origin, from having met with cancer in other parts of the skeletons in which it occurred. This tumor must not be confounded with those enlargements of the bone which are produced by an accumulation of blood, owing to rupture of the bloodvessels and consequent hemorrhage into the cancelli, or between the periosteum and the bone. Cruveilhier, who inclines to the same view as Rokitansky, gives delineations[1] of a remarkable case, in which a lady, æt. thirty-eight, of a good constitution, and without hereditary taint, the mother of eight children, presented a dozen tumors of the size of a walnut, situated on the head. They were soft, and pulsated; the beats were isochronous with that of the pulse, and were accompanied with a blowing noise, similar to one heard at the aorta. There were several similar tumors in other parts of the body, but those of the head only proved after death to belong to the bones. They exhibited on section a filamentous areolar structure filled with blood; the destruction of the bone penetrated to the dura mater; the absorption of the osseous tissue resembled that produced by aneurism; on the external surface there was evidence of an attempt at repair, in the shape of osseous vegetations. Cruveilhier is of opinion that in this case the vascular development took place mainly at the ex-

[1] Anatomie Pathologique, tom. ii. livr. xxxiii. pl. iv.

pense of the arterial system; he thence infers a general law that there are two kinds of erectile tumor, one of a venous, the other of an arterial character. He regards the latter as analogous with fungus hæmatodes.

Mr. Stanley describes a sanguineous tumor of bone, always originating in the cancellous texture, by blood being effused into it, and causing a gradual enlargement of the cells and absorption of the septa. The walls of the bone are thus gradually expanded into a globular cyst of varying thickness and extent. According to the stage of the disease the blood is found in cells, intersected by fibres or laminæ and fibres, the remains of the original fabric of the bone; or, in a more advanced stage, in a single cyst. A feeling of fluctuation may be thus produced, and gradual ulceration may give rise to a discharge externally. These tumors occur chiefly in the articular ends of bones, and most frequently within the condyles of the femur or head of the tibia. A form of sanguineous tumor of the head, met with in infancy as a result of the pressure exerted upon the cranial bones during parturition, and known by the term cephalhæmatoma, has given rise to much discussion, as the symptoms have been variously explained by different observers.[1]

It consists of an effusion of blood between the pericranium and the bone, and is most commonly met with on one of the parietal bones. Rare cases are recorded of an internal cephalhæmatoma, in which the extravasation took place between the dura mater and the bone. All authors are agreed that external cephalhæmatoma occurs most frequently on the right side. Bednar (quoted by Mr. Willshire) found, that of 74 examples, 40 were on the right, 22 on the left, 6 over each parietal bone, 4 on the occipital, 1 over both parietals, and the occipital, and 1 over the frontal bone, the latter being the smallest.

Older writers have regarded it as depending upon an essential disease of the bone, owing to the hard ring bounding the tumor, and inducing the impression that the bone has been excavated. A considerable difference of opinion still exists as to the exact rationale of its production, and more particularly as to the point of the maternal organs at which the pressure is effected that produces the tumor; but most observers agree as to its being immediately connected with hemorrhage from the torn vessels; the blood coagulates, and a reparative process is set up, which causes a fibrinous pad at the circumference, which, in its turn, may ossify, if absorption does not ensue rapidly. Whether or not a diseased state of the fœtal bloodvessels may predispose to the affection, is a question which is answered in the affirmative by some, while it is denied by others; it is certain that the chief argument of a primary disease of the bone is destroyed by the more careful analysis of cases by recent observers; showing the ring of bone either not to exist at all, or else to be a secondary ossification of the coagulum, accompanied by an inflammatory process. This, according to Rokitansky, commences at the margin of the denuded part, and produces the deposit of bony matter in the form of a velvety or finely filamentous osteophyte. It appears that

[1] A good *résumé* of their opinions is given by Dr. Willshire, in the tenth volume of the British and Foreign Medico-Chirurgical Review, July, 1852, p. 6.

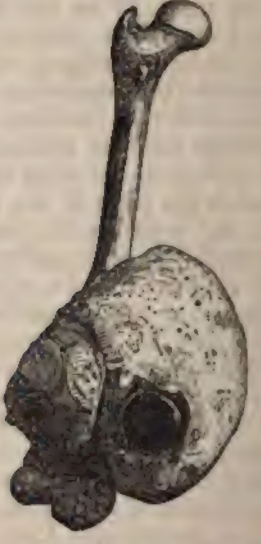
artificial interference commonly becomes necessary for the removal of the coagula, suppurative inflammation being otherwise liable to set in and give rise to carious destruction of the bone.

CYSTS.

Cysts occur very rarely in bone. A very remarkable instance is described by Mr. Keate,[1] in which a large tumor in the frontal bone of a young woman, æt. 18, was formed by the development of hydatids between the plates of the bone. Frequent attempts to destroy the cyst and its contents, by escharotics, after removal of a portion of the external osseous sheath, failed; and the constant sprouting out of fresh hydatids at last induced Mr. Keate to saw off the entire tumor, after which the girl completely recovered. The diameters of the exposed surface were four and a half by four inches. In the very compact and hard bony substance, forming the base of the tumor, were five or six cells containing hydatid cysts. It appears that from these cells the hydatids were constantly regenerated, forcing their way into the large cavity of the tumor, and yielding to no treatment, as the remedies failed to destroy the matrix from which they pullulated.

Mr. Stanley quotes some cases from his own experience, and that of

Fig. 395.



Osteocystoma, of large size; occupying lower end of femur.—Prep. in University Museum.

other observers, from which it appears that the bones of the skeleton are all equally liable to be attacked. The development of hydatids

[1] Medico-Chirurgical Transactions, vol. x. p. 278.

induces a gradual, painless expansion of the bone, which may thus become perforated and allow of an escape of the hydatids. The cavity of the bone in which the hydatids form is lined by an adventitious cyst, and this is said to be liable to excite inflammation in the surrounding bone, as well as to induce purulent products in the cysts. Cysts of this description often contain matter, to the naked eye, absolutely identical with laudable pus, which, however, under the microscope, exhibits none of the characters of pus. We have recently examined two hydatid cysts of this kind, removed from the livers of two patients of St. Mary's Hospital, on which the bright green, semifluid contents showed no trace of pus, but granular matter, granular corpuscles, varying much in size and shape, the granules being highly refracting particles of oil, much greenish-colored oil and echinococci, entire or in parts. Had these cysts been found to contain pus, where there was no trace of inflammatory action in the vicinity, it would have been difficult to reconcile its presence with the usual theory of suppuration, the more so as the cyst envelop cannot be shown to contain any vessels. It is probable, therefore, that, in those cases in which bone hydatids present puriform contents, these must be interpreted according to the views suggested by the above observations.

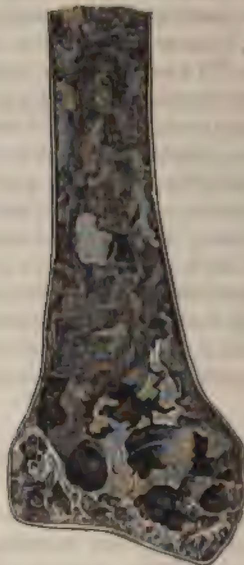
MOLLITIES OSSIUM.

Mollities ossium, osteomalacia, or malacosteon, is a disease regarding which the views of authors differ; some treating it as essentially distinct from other known osseous maladies, some as a form of atrophy; others, again, as identical with rachitis, except in that it attacks adults instead of children. The rarity of the disease is one great obstacle to our arriving at a satisfactory conclusion; but, so far as the evidence reaches that we have been able to examine, there appears to be every reason for regarding it as much deserving of a separate place in pathology as any disease of the bones which we have investigated.

The disease consists, as the term indicates, essentially in a softening of the bones, brought about by an absorption of the earthy matter, and the substitution of a large quantity of fat. It is an entire perversion of the process of nutrition, in as far as regards the skeleton, the earthy phosphates being eliminated from the system by the kidneys, while a deposit of fat takes place into the cartilaginous matrix, a process which necessarily induces great pliability and fragility of the bones. As the bones of the trunk are especially liable to be attacked, the individual affected becomes reduced in size from the collapse of the vertebral column. A tall subject may thus be converted into a dwarf; an instance of which we had an occasion of seeing in the Clinique of Professor Kilian, of Bonn. The individual was a married woman, whose stature had diminished in this manner, and was doubly interesting from having in this condition become the subject of a successful Cæsarean operation. It was subsequent to her recovery from this ordeal that we saw her, and that she was supposed to be again pregnant. Dr. Greenhalgh enabled us recently to examine a similar instance, in which the disease was also followed by contraction of the pelvis, rendering the Cæsarean section

necessary. Adults, and especially females, are the subjects of the malady. It attacks women chiefly after they have commenced child-bearing. Mr. Curling[1] has collected sixteen cases, thirteen of which

Fig. 396.



Section of a femur from a lady æt. 30, affected for some years with mollities ossium. The walls of the bone are thin, soft, and flexible: the place of its medullary and cancellous tissue is occupied by soft, jelly-like, transparent fat, of various shades of yellow and pink, some of it deep crimson; a similar kind of fat appeared to be diffused through the proper texture of the walls.—St. Bartholomew's Museum, i. 233.

occurred in females, and three in males. Eleven were fatal between thirty and forty. In none it showed itself before puberty; but two patients were above fifty years of age. Several of them were delivered of children during the progress of the complaint. It is not associated with any particular lesion of the viscera; neither can an hereditary or idiopathic taint, or diathesis, be traced, by which the peculiar symptoms could be explained or referred to a known type. Kilian[2] treats of mollities ossium as presenting two varieties—the waxy (cerea), and the fragile (fracturosa). In the former, the bones generally, but especially those of the pelvis, present a dirty, dark-yellow color. They lose their transparency in the middle, while their weight is not much diminished, and they become flexible, like wax; in the second, the bones present a snowy whiteness, and a light transparent, open texture, rendering the bones so fragile that they give way under the mere pressure of the finger. The first kind of bones do not dry clean, but remain greasy; the second dry quickly, and give no greasy feel. Both varieties, according to Kilian, exert the same influence upon the pelvis in regard to

[1] Medico-Chirurg. Trans. vol. xx. p. 336.

[2] Kilian, H. F. Beitrag zu einer genaueren Kenntniss der allgemeinen Knochenerweichung, &c., Bonn, 1829; and Die Geburtslehre, &c., vol. ii. p. 367, 1840.

the distortions that are produced. These he describes as consisting in angular deflections of the individual bones, and a mutual approximation of the bones in the conjugate and transverse diameters. If Professor Kilian's view regarding the two species of the malady be correct, it is probable that they would be distinguished by their chemical constitution, and this may be assumed as the reason why the chemical analysis of the bones affected with osteo-malacia have yielded results so widely apart. Thus, in one of the two remarkable cases detailed by Mr. Solly,[1] the analysis of the affected bone, by Dr. Leeson, showed 100 parts to contain

Animal matter	18.75
Phosphate and carbonate of lime	29.17
Water	52.08
	100.00

The chemical examination of a case (detailed by Dr. Ramsbotham, in the Reports of the Pathological Society)[2] by Dr. Garrod, yielded,

Fatty matter	20.35
Gelatin yielding matter	58.37
Carbonate and phosphate of lime, and phosphate of magnesia	21.28
	100.00

In the former analysis, we see nearly eight parts more earthy matter than in the latter; nor can it be objected that the analyses were made at different stages of the disease, because, in both instances, the patients from whom the specimens were taken had succumbed to the malady. The analysis given by Dr. Bostock,[3] reduces the amount of earthy matter to a yet lower figure. In a specimen that he examined, he found the proportions to be, in one hundred parts of bone:—

Jelly and oil	22.5
Cartilage	57.25
Earthy matter	20.25

Mr. Stanley quotes an analysis of Dr. Bostock's, in which the constitution of the entire bone was, in 100 parts of oil, about 67; of membrane, about 20; of earthy salts, about 11.

The microscope shows the structure of the bone altered; the corpuscles and their canaliculi having lost their earthy contents, are empty and transparent, and only faintly visible; while the Haversian canals are unnaturally enlarged. Dr. Hall Davis, in his report of the microscopic appearances of the case of Dr. Ramsbotham, above quoted, states that, besides fat and blood, he found nucleated cells. Rokitansky appears to consider it allied to malignant disease, but does not mention the presence of compound corpuscles; it therefore yet remains to determine, as far as the microscope can do it, the relation to cancer; and it seems probable that, while we may thus establish a true fatty degeneration of bone, we may also prove that many cases of mollities ossium

[1] Medico-Chirurgical Transactions, vol. xxvii. p. 435.
[2] Reports, &c., 1847–48.
[3] Medico-Chirurgical Transactions, vol. iv. p. 38.

Fig. 397.

Bone-corpuscles; *a*, in the normal state; *b*, enlarged, as in mollities ossium.—Dalrymple.

are essentially primary cancer of the skeleton. We have ourselves spoken of a case of this kind (p. 712).

In a well-marked instance of mollities ossium, of which the history was given to the Medico-Chirurgical Society by Dr. T. K. Chambers,[1] and which occurred in a female æt. twenty-six, the bones throughout the system were soft and yielding, so that a sharp knife could readily pass through them. A portion submitted to our examination, which was removed from the tibia, resembled rather a piece of muscle than of bone. The periosteum was entire, and it was only in connection with it that a few minute spiculæ of bone could be found. In these, there was a faint trace of the bone-corpuscles, but they were filled with reddish oil. The remaining tissue consisted of large, transparent oil-vesicles, and minute globules of reddish oil. The muscular tissue examined, though to the naked eye healthy, was entirely deprived of its normal structure, and converted into reddish corpuscles, of from $\frac{2-3}{10000}$ of an inch in diameter, intermingled with large oil-vesicles. We saw nothing at all resembling the character of malignant growth.

THE MEDULLA.

The medulla undoubtedly participates in the nutritive and morbid processes affecting the bones individually or generally; and we have already had occasion to see that, in several of the diseases of the latter, changes in the medulla form an essential constituent of the malady; as in encephaloid cancer, or mollities ossium. It is yet to be determined in how far the medulla is liable to become primarily affected. It varies in consistency according to the vigor of the individual; while in dropsical and phthisical cases we find it thin and serous, or yellow in icterus, or very scanty in ivory condensation of a bone; it exhibits greater firmness, and a richer pink hue, in habits tending to an inflammatory character. Lobstein states that he has repeatedly verified the existence of inflammation of the medulla, but invariably associated with inflammation of the reticular tissue of the bone. Its color, in this case, resembled that of kermes, and its density that of fibrin. He observed it not only in the medulla contained in the diaphyses, but also in the epiphyses of cylindrical, in the diploe of flat bones, and in the reticular substance of certain bones at the base of the cranium, such as the basilar

[1] Medical Times and Gazette, March 25, 1854.

on of the occipital and of the body of the sphenoid. In the latter , he states that he has met with this condition more frequently in any other region; a remark that deserves attention in reference ertain observations of other authors regarding the constriction of 'oramen magnum in producing epilepsy, and the enlargement of the toid process in connection with chorea. The real seat of inflamon in bone, as Rokitansky remarks, is the membrane that lines its ies; it is, therefore, fair to infer that, in all diseases dependent the state of the vascular system, whether of an ordinary or of a gnant character, the medulla is affected coincidently with, if not iously to, the bony tissue itself.

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